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CORTISONE AND ANÆSTHESIA.[1]

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IT is now nearly seven years since cortisone and allied drugs were first introduced into therapeutics. In more recent years, enthusiasm has been tempered by the realization that there are definite indications for these drugs and—more important—definite contraindications to their use. It is now recognized that the use of cortisone and allied compounds may result in serious complications, some of which are of particular interest to surgeon and anæsthetist.

Cortisone is now being employed in pre-operative preparation for certain types of operation and, in addition, patients who have received cortisone for some medical condition not infrequently come to anæsthesia and operation. The anæsthetist must, therefore, be aware of the physiological effects of these hormones, of their uses in medicine and surgery, and of their dangers—particularly in the operative and post-operative periods.

PHYSIOLOGY.

Steroid hormones are liberated by the adrenal cortex, which is activated by the pituitary hormone, corticotrophin (ACTH). Numerous steroids have been isolated from the adrenal cortex; the two synthetic preparations that have been used therapeutically are cortisone and hydrocortisone. Exogenous cortisone is probably changed in the body to hydrocortisone and has a similar action. In most instances the two agents are interchangeable. ACTH liberates other hormones as well as hydrocortisone, including androgens. Therefore the biological effects of ACTH, although essentially similar, differ to some extent from those produced by the administration of cortisone and hydrocortisone.

The indications for ACTH, cortisone and hydrocortisone are the same, except that cortisone, and not ACTH, is commonly used in adrenal insufficiency or severe hypopituitarism. ACTH is useful in reactivating adrenals that have been suppressed with cortisone or hydrocortisone, but it must be remembered that this reactivation may take five days or longer to occur.

SIDE EFFECTS OF CORTISONE.

Cortisone is necessary for life, but when given in large doses may produce dangerous side effects which are of particular interest to the anæsthetist. These side effects include the following:

1. Adrenal suppression. This may result in acute adrenal insufficiency if cortisone or ACTH is suddenly withdrawn.

2. Electrolyte disturbances. Sodium retention results in œdema and hypertension, and potassium excretion is increased, with resulting muscle weakness, distension and abdominal pains.

[1] Read at the annual meeting of the Australian Society of Anæsthetists in April, 1956, at Melbourne.

3. Cushing-like effects. Obesity of the trunk and "mooning" of the face are frequently described.

4. Hyperglycæmia and glycosuria. These are not very important except in a diabetic patient, to whom extra insulin should be given.

5. Anti-inflammatory action. This prevents mobilization of cellular reaction to infection, which is of serious consequence in many infections. Cortisone may mask pain and shock in such conditions as acute peritonitis, and may permit reactivation of a tuberculous focus.

6. Retardation of wound healing. This is probably due to the impairment of fibrinoplasia.

7. Psychic changes. These are most important, and are likely to occur particularly in patients with a background of nervous instability. Insomnia is common.

8. Peptic ulceration. Silent perforation of a peptic ulcer has been reported as having occurred in a number of patients receiving cortisone.

9. Venous thrombosis. An increased tendency to thrombosis in the calf veins, and also in the portal vein, has been described (Adlersberg, Stricker and Himes, 1955).

10. Fatty liver. In rats cortisone increases the susceptibility of the liver to toxic agents, including chloroform (Aterman and Ahmad, 1953).

Cortisone in Surgical Cases.

Three main classes of surgical patients receive cortisone: (i) patients receiving cortisone in physiological doses as substitution therapy; (ii) patients receiving cortisone as a therapeutic measure, either for the surgical condition or for some incidental disease; (iii) patients who are not receiving cortisone at the time of operation, but who have previously been treated with the hormone.

Cortisone in Replacement Therapy.

Cortisone is used in small physiological doses as replacement therapy in patients suffering from pituitary or adrenal hypofunction, and in the preparation for pituitary and adrenal operation. Included in this group are the following operations: (*a*) Operative procedures on patients with Addison's disease or acute adrenal insufficiency. (*b*) Bilateral adrenalectomy for inoperable carcinoma. (*c*) Unilateral adrenalectomy for a hyperfunctioning adrenal—for example, phæochromocytoma. In these cases the other adrenal is atrophic, and cortisone must be given until the normal adrenal commences to function. (*d*) Pituitary surgery, including removal of pituitary tumours and hypophysectomy.

In the preparation of such patients, there are various regimes which may be followed. Two of them are as follows:

1. Pre-operative doses of 200 milligrammes may be given by intramuscular injection forty-eight hours, twenty-four hours and two hours before operation. Post-operatively, 50 milligrammes are given intramuscularly six-hourly for twenty-four hours, and then gradually decreasing oral doses are given over the next five days until a small maintenance dose can be achieved.

2. An alternative method, which is sometimes used in major pituitary surgery such as hypophysectomy, is to administer 100 milligrammes of cortisone the night before operation and again two hours before operation. A continuous intravenous drip administration of hydrocortisone at a rate of 12 to 25 milligrammes per hour is maintained throughout the operation and for the first twelve to twenty-four hours afterwards until oral cortisone therapy can be commenced.

Cortisone in Medical Conditions.

The anæsthetist should be aware of the variety of unrelated conditions in which benefit is derived from the administration of cortisone. In contradistinction to its use in replacement therapy, large doses must often be employed in alleviating medical conditions, and therefore side effects are more likely to occur.

The hormone may be employed systemically in collagen diseases, bronchial asthma, skin conditions, blood dyscrasias and diseases of the alimentary tract, notably ulcerative colitis. Most commonly the anæsthetist will meet rheumatoid patients who are to undergo orthopædic procedures, patients with ulcerative colitis who are to have major abdominal operations, patients with thrombocytopenic purpura who require splenectomy and asthmatics who are to have some form of surgical intervention.

Cortisone and the Anæsthetist.

There are three features in particular which concern the anæsthetist when the patient is receiving cortisone: (i) the prevention, recognition and management of acute adrenal insufficiency; (ii) the choice of anæsthetic agent; (iii) the masking of shock.

Acute Adrenal Insufficiency.

During operation and anæsthesia, even of the most minor nature, the adrenal cortex is stimulated via the pituitary to increase secretions of steroid hormones. Biochemical studies show that adrenal cortical stimulation commences during or shortly after operation, and continues for one to three days (Hume, 1953; Franksson, Gemzell and von Euler, 1954; Tyler, Sandberg and Eik-Nes, 1952). Patients suffering from disease of the pituitary or adrenal glands or those whose adrenals have been suppressed by the administration of adreno-cortical hormones are likely to develop acute adrenal insufficiency under stress. Experimental studies indicate that surgical trauma, especially if prolonged, makes greater demand on the adrenal cortex than does anæsthesia or hypoxia.

There have been a number of reports of post-operative deaths from acute adrenal insufficiency associated with functional atrophy induced by cortisone administration (Lewis *et alii*, 1953; Salassa *et alii*, 1953; Fraser *et alii*, 1952).

From the Mayo Clinic, Salassa *et alii* present two case reports in which patients, who had previously received cortisone, developed acute adrenal insufficiency in the early post-operative period. One patient underwent gastrectomy and the other bunionectomy, and in both cases operation and anæsthesia were uneventful, but the patients died within a few hours in irreversible shock. *Post mortem*, atrophy of the adrenal cortex was found on microscopic examination.

Fraser (1952) reports the occurrence of acute adrenal insufficiency after a cup arthroplasty; this patient had not received cortisone for four and a half months prior to operation. Thus it appears that atrophy may persist for a long time after the withdrawal of cortisone.

The following is a case report from the Royal Melbourne Hospital.

A woman, aged sixty-two years, was admitted to the Royal Melbourne Hospital on June 8, 1955, suffering from severe ulcerative colitis. Intensive medical treatment had included intravenous therapy, blood transfusion, and the administration of antibiotics and cortisone. For the first twelve days her progress was variable, but on the whole she showed improvement, although some abdominal distension persisted. At the same time she had some œdema of the sacrum and legs, and there was fluid at the bases of her lung.

On June 22, twelve days after her admission to hospital, she began to deteriorate rapidly, and by the morning of June 23 she was vomiting fæculent material. At this stage she was in too severe a state of shock for operation, but intravenous therapy with saline and albumin improved her condition, and she was brought to the operating theatre on the evening of June 23.

In the anæsthetic room she was sweating, cyanosed and dyspnœic, with a pulse rate of 120 per minute and a blood pressure of 240 millimetres of mercury, systolic, and 130 millimetres, diastolic. Much of this respiratory distress was due to her severe abdominal distension, and her condition improved considerably after the induction of anæsthesia with thiopentone and *d*-tubocurarine and the associated inflation of the lungs with oxygen.

At laparotomy she was found to have severe peritonitis and three separate perforations of the colon. Surgical pro-

cedures performed were terminal ileostomy, total colectomy, and proctosigmoidectomy. Throughout these procedures she maintained her condition remarkably well, and she was conscious at the end of the operation.

Her post-operative condition was most satisfactory for twenty-six hours, when she suddenly went into extreme peripheral circulatory failure and lost consciousness. Blood transfusion, pressor agents and artificial respiration with oxygen were of no avail, and she died within fifteen minutes.

At autopsy her heart was found to be normal, but there was severe congestion of the lungs with some collapse of the right lung. In the abdomen there was obvious evidence of peritonitis. Macroscopic examination of the adrenal glands showed them to be of normal size, with some lipoid vesicles in the cortex. On microscopic examination, the adrenal glands were found to be œdematous, and in the cortex there were many degenerative and atrophic cells. These changes were patchy, but resulted in some thinning of the cortex.

This patient presents a typical history of acute adrenal insufficiency with sudden irreversible circulatory collapse occurring twenty-six hours after operation. For thirteen days prior to operation she received a total of 1375 milligrammes of cortisone and was on a daily maintenance dose of 50 milligrammes. On the day of her operation her dose was not increased and she received only 25 milligrammes, owing to the fact that her second dose would have been administered at a time when she was in the operating theatre. After operation she received 50 milligrammes intramuscularly. This dosage is considered to have provided inadequate coverage for what proved to be a traumatizing operation, and in addition this patient had already shown some signs of hypercortisonism (œdema, moon facies), from which it could be suspected that adrenal atrophy was present. Admittedly this patient was extremely ill with peritonitis following multiple perforations of the colon and had undergone a prolonged operation. In such a patient even a minor degree of adrenal insufficiency might have been lethal. However, the supposition from clinical evidence that acute adrenal insufficiency contributed to the death of this patient is upheld by the microscopic appearance of the adrenal cortex.

Only a small percentage of patients who receive cortisone will develop acute adrenal insufficiency following surgical trauma, but it is important that the anæsthetist and surgeon should be able to assess and treat pre-operatively patients who are likely to develop adrenal insufficiency, and that they should be able to diagnose and treat the condition immediately.

Pre-Operative Assessment and Management.

The following are guides to the recognition of the possibility of acute adrenal insufficiency.

1. A history of cortisone administration, especially if prolonged, within the preceding six months. Unfortunately this information is not always available; but if the patient has suffered from any of the medical conditions mentioned earlier it should be assumed that he may have received cortisone and more accurate information should be obtained. The patient receiving high maintenance doses should be regarded as being vulnerable to acute adrenal insufficiency. In this connexion it is important that the type of cortisone be known. Recently there has been introduced a preparation, prednisone (the unsaturated analogue of cortisone), of which the dose is one-third to one-fifth that of cortisone or hydrocortisone. The anæsthetist must be sure that what appears to be a small maintenance dose of cortisone is not, in fact, a relatively high dose of prednisone.

It is not certain how long a course of cortisone is necessary before adrenal atrophy may occur, nor how long after withdrawal it may persist. Salassa *et alii* (1953) sugggest that any patient who has received as little as five days' treatment with cortisone within the previous six months is capable of developing adrenal insufficiency.

2. Signs of hypercortisonism or a history of such signs should put the anæsthetist on the alert.

3. Adrenal function tests. In non-urgent cases it may be useful to assess adrenal function by various tests, but these may not be completely reliable. Estimations of the serum electrolyte contents are useful, and Perlmutter has described a relatively rapid method of assessing adrenal function by measuring both plasma and urinary sodium levels. If adrenal function is impaired, the plasma sodium level is low while urinary sodium secretion is high, owing to inability of the tubules to reabsorb sodium in the absence of adrenal cortical hormones.

Management of Patients Receiving Cortisone.—The usual management of patients receiving cortisone is to administer large doses of cortisone twenty-four hours and two hours before operation. The actual dose depends on the duration of previous cortisone treatment and on the severity of the operation. Usually 100 to 200 milligrammes are given by intramuscular injection. The cortisone should be given in gradually decreasing doses for three to four days after operation. ACTH is useful only when there is plenty of time for preparation. The minimum time required is three to five days.

If the patient is already receiving cortisone at the time of operation the dose should be increased and additional cortisone should be given by intramuscular injection twenty-four hours and two hours before operation.

Care should be taken to ensure that administration of the hormone is continued, if possible orally, immediately after the patient's return from the operating theatre. It is in the early post-operative period that the danger of insufficiency is greatest, and it is in this period that administration of cortisone is most likely to be overlooked. Whenever possible an endocrinologist or physician should be consulted about the management of these patients.

Signs of Acute Adrenal Insufficiency.—Acute adrenal insufficiency usually develops in the first forty-eight hours of the post-operative period, although it may occur during operation. The most typical picture is one of sudden and extreme circulatory collapse which does not respond to the usual methods of resuscitation, including transfusions and the administration of pressor drugs. Mental confusion, coma and hyperpyrexia may also be apparent. Unfortunately circulatory collapse may be so sudden that there is little time to combat it; therefore when any patient develops unexplained or unexpected circulatory collapse, adrenal insufficiency should be considered.

The Management of Acute Adrenal Insufficiency.—The management of acute adrenal insufficiency consists of immediate intravenous transfusion with hydrocortisone in a strength of 100 milligrammes in 500 cubic centimetres of saline. This should be administered by the drip method at a rate of 12 to 25 milligrammes per hour. In addition, when hypotension persists a vasopressor such as l-noradrenaline may be added to the solution.

While it is outside the scope of this paper to discuss the difficult problem of electrolyte balance in acute adrenal insufficiency, it should be noted that the intravenous administration of large quantities of saline and glucose must be avoided and attention must be paid to the possibility of potassium deficiency.

Owing to the very rapid collapse of these patients there should be no delay in instituting cortisone therapy, but if posible an endocrinologist should be consulted with regard to further treatment.

Choice of Anæsthesia for Patients Receiving Cortisone.

There are three factors which should influence the anæsthetist in his choice of anæsthetic agent.

1. The main factor is the danger of precipitating acute adrenal insufficiency. Stress, particularly the stress of surgical trauma, is known to precipitate adrenal cortical insufficiency. But it is uncertain how much demand anæsthesia and hypoxia make on the adrenal cortex. Thorn *et alii* (1953) were unable to produce measurable adrenal stimulation with severe hypoxia, cold, strenuous exercise of psychomotor tests. However, it is advisable to employ techniques in which excitement and sympathetic stimulation are at a minimum, and hypoxia must always be avoided. In addition, patients with adrenal cortical insufficiency are particularly sensitive to all analgesic and anæsthetic drugs, especially barbiturates and opium alkaloids.

2. The second factor is that cortisone causes hyperglycæmia and glycosuria. While this is not serious, except in diabetics, it is advisable to avoid anæsthetic agents such as ether which have the same effect.

3. There is some evidence that cortisone causes deposition of liver fat and increases the susceptibility of the liver to toxic agents such as chloroform. Therefore agents which spare the liver as much as possible should be employed.

In anæsthesia for patients receiving cortisone, no preoperative sedation with barbiturates, opium alkaloids or pethidine should be given. Thiopentone should be omitted or given in minimal doses, and analgesia should be obtained by an inhalation agent such as nitrous oxide or cyclopropane, but ether should be avoided. Relaxation can be effected with any of the common relaxant agents, but dosage should also be at a minimum. Hypoxia must be avoided at all times, and for this reason cyclopropane appears better than nitrous oxide, as better oxygenation is possible. The employment of cyclopropane also considerably reduces the amount of relaxant required.

Cortisone and the Masking of Shock.

Cortisone mitigates the outward manifestations of disease, and a number of cases have been reported in which perforation of a viscus has not been accompanied by evidence of shock or peritonitis until a very late stage. The patient whose case was reported above had multiple perforations of the bowel, but did not show signs of undue distress until the associated abdominal distension caused respiratory embarrassment. It is therefore important that the anæsthetist should realize that patients receiving cortisone may have a more severe lesion than is apparent, and that they should thus never be taken lightly, nor should undue blood loss be allowed to occur.

It has been shown that blood vessels constrict adequately only in the presence of adrenal cortical hormones (Zweifach, 1951). If the patient is suffering from an insufficiency of these hormones, it is possible that considerable capillary oozing may occur. This may be responsible for one case of pituitary tumour encountered recently, in which adequate hæmostasis was impossible for some hours after the surgical removal of the tumour.

Conclusion.

Cortisone has a definite place in modern therapeutics, both as replacement therapy and in the empirical treatment of a large variety of conditions. However, there is considerable evidence that cortisone may cause adrenal atrophy, and that this effect may outlast the treatment for some considerable time. A case of severe ulcerative colitis is reported in which signs of acute adrenal insufficiency developed after a major operation. The patient had received cortisone prior to operation and apparently inadequate cortisone dosage after operation.

It is therefore important to consider carefully the possibility of adrenal cortical insufficiency in relation to any surgical patient who has received cortisone, hydrocortisone or ACTH. When there has been prolonged treatment or evidence of hypercortisonism at any stage, these patients should before operation be treated prophylactically with cortisone.

Whether or not these patients have received prophylactic treatment, they must be treated with great care with regard to pre-operative preparation, choice of anæsthetic, degree of surgical trauma, assessment of shock and post-operative supervision.

Only a small percentage of patients receiving cortisone are likely to develop adrenal cortical insufficiency, and it is not always possible to foresee which type of patient this will be. The anæsthetist, therefore, must exert constant vigilance in all cases so that unnecessary operative and post-operative deaths do not occur.

Acknowledgements.

I am indebted to Dr. Ian Wood and Dr. E. S. R. Hughes for permission to publish the case report of their patient, and to Dr. J. D. Hicks for the autopsy report. I also wish to acknowledge the invaluable assistance given me in the preparation of this paper by Dr. I. Wood and Dr. H. P. Taft.

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THE AGGRESSIVE TREATMENT OF METASTASES.[1]

By Edward Wilson,
Sydney.

In recent years there has been a change in the attitude of medical practice. Instead of a fatalistic acceptance of the presence of metastases in particular patients, more aggressive medical treatment is now the order of the day. With the introduction of supraradical surgery, second-look procedures, multiple viscera resection, hormone therapy, adrenalectomy, *et cetera*, the doldrums of previous decades in the treatment of metastases are gone. Improvements in anæsthesia, the introduction of chemotherapy, and a better understanding of metabolic requirements have all aided progress in the treatment of metastases. Advances have also been made in surgical technique, including such procedures as the replacement of defects in large arteries by venous or frozen arterial grafts, and the newer methods of uretero-sigmoid anastomosis.

Our knowledge of the very early detection of metastases is non-existent. For only a few types of metastases are there diagnostic tests, and even these are not infallible. The tests, some of which are listed in Table I, are based on the recognition of secretion products or on the changes in the host due to indirect effects. The use of radioactive isotopes for the diagnosis and the localization of metastases may have a great future, but at present it is still experimental. A procedure which may help in the recognition of metastases is that of the vital staining following the injection of a dye (direct sky blue) near a primary growth. This dye produces a blue staining of the lymph channels and nodes except where blocked or replaced by cancer cells. Other methods of diagnosis, such as the cytological examination of surface cells (Papanicolaou), are of more value in the recognition of the primary growth than in that of the metastases. For the very early diagnosis of metastases little real help is to be expected from any of these methods, and we are therefore usually left with our clinical acumen.

[1] Read at a meeting of the Section of Surgery, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

Hopes have always been high that the problem of the cause of cancer might be solved and that metastases might be prevented; but, excepting by the early ablation of the primary growth and adjacent lymphatic field, our means of preventing metastases is limited. For years, the avoidance of massage or manipulation of a tumour has, or should have been, practised. The danger of such handling has been clearly demonstrated by Turnbull and Fisher (1954). Ligation of the vascular pedicle is already widely used at an early stage in operations upon malignant tumours of the kidney and of the testis, but its use should be extended as far as possible to operations for other cancers.

TABLE I.

Tests for the Detection of Metastases.

Test.	Origin of Metastases.
Serum acid phosphatase estimation.	Carcinoma of prostate.
Serum alkaline phosphatase estimation.	Metastases in bone.
Serum calcium and phosphorus estimation.	Carcinoma of parathyroid.
Urinary 17-ketosteroid excretion.	Carcinoma of adrenal cortex.
Urinary gonadotropin excretion.	Some testicular tumours; chorion-epithelioma.
Urinary œstrogen excretion.	Feminizing adrenal cortical tumour.
Estimation of basal metabolic rate.	Carcinoma of thyroid.
Presence of atypical protein in blood and urine.	Multiple myeloma.
Chemical changes in pleural or peritoneal exudate.	Metastases in pleura or peritoneum.

Numerous futile efforts have been made to delay the growth of metastases, as for example by restriction of the caloric intake. Instead of delaying their growth, it would be a greater advance if cancer cells could be stimulated to develop the characteristics of normal cells. Microscopically, such a change would be similar to that produced by liver extracts *et cetera* in the bone marrow of the patient with pernicious anæmia when on treatment the change from an abnormal to a normal cell type occurs. In pernicious anæmia it is to be noted that the return to normal with liver therapy is not a permanent change, for a reversion to the original abnormal cell type is held in abeyance only by the continuation of therapy. Even so, more study of the effects of substances which stimulate the growth or metabolism of normal cells would seem to be indicated.

Thyroid extract has been used to treat metastases by Loeser (1953), who stated that hyperthyroid patients develop cancer to a lesser degree than hypothyroid ones. However, in seven patients with advanced metastases I was unable to detect any benefit from thyroid administration. Further, in patients suffering from carcinoma of the thyroid gland there does not appear to be any relationship between the presence of thyrotoxicosis and the prognosis. It is of interest that the prognosis is said (Dargent, 1949) to be worse in patients who develop a mammary carcinoma after bilateral oophorectomy has already been performed. May not this be due to a deficiency of œstrogens which usually assist in the attainment of maturity of the breast cells?

The rate of growth of some metastases is slow, possibly because of local tissue resistance. Growth may even be discontinuous, that is, the cells apparently pass into a dormant state, which, Hadfield suggests, is due to a temporary mitotic arrest. In a recent paper Morton and Morton (1953) collected a series of cases which indicates that cancer may behave as a chronic disease. Four out of their 17 patients had melanomata. I have had one patient from whom several metastatic malignant melanomata were removed from the subcutaneous tissues more than twenty years after metastases had first been removed. A very slow rate of growth is also occasionally seen when there are epitheliomatous metastases in the cervical lymph nodes. For example, a patient of mine, who had an epithelioma of the tongue removed eighteen years ago, developed a lump in the neck which was found to contain metastatic epithelioma. No other primary tumour or metastasis was found. This story could be repeated many times.

The failure of many malignant emboli to thrive in the lungs and other viscera was demonstrated by Schmidt. It is because of a fear that the hormone changes of pregnancy will stimulate such dormant metastases from a mammary carcinoma that termination of pregnancy is often advised. One point of interest here is that metastases which have remained microscopic are commonly found in the ovaries and adrenal glands of patients submitted to oophorectomy and adrenalectomy for the palliation of advanced carcinoma. Occasionally, such adrenal metastases progress and temporary improvement in the general condition occurs, only to be followed by death from adrenal failure (Slaughter, 1953). Why certain malignant growths, for example, *spongioblastoma multiforme*, do not metastasize is obscure. We might hazard a guess that the typically thickened blood vessels of this growth are resistant to invasion; and we might even hazard a further guess that the delayed appearance of metastases of most cancers in the elderly has a similar basis.

Treatment.

Apart from such theorizing, there are ways of interfering with the growth of some metastases once they have appeared. Such methods are surgery, irradiation and hormone therapy; and of these surgical attack is still the chief. Radiotherapy has an honoured place in the treatment of cancer, but with subcaustic doses the response of metastases differs greatly. The use of radioactive isotopes for the treatment of cancer by intravenous or interstitial injection is still in the experimental stage.

Hormone Therapy.

Other methods include the use of hormones or operations to alter the hormone environment of the metastases; but, although it may well be that the future treatment of metastases will lie more with hormonal methods, the present uses of the latter are only prophylactic and palliative. Some mammary and prostatic carcinomata are demonstrably affected by hormones. Other cancers, such as chorion carcinoma, malignant melanoma, and adenocarcinoma of the uterus, are probably hormone dependent, but so far hormone treatment of these cancers has met with little success. In assessing the results of hormone therapy, its non-specific effects such as improved protein synthesis *et cetera* are to be remembered.

Prophylactic hormone treatment is an attempt to modify the development of metastases by the administration of hormones, or by operations such as adrenalectomy and oophorectomy, before any distant metastases are detected. An attempt is thus being made to deal with a problem which has previously been ignored, but its value is still not proven. Palliative hormone treatment involves the administration of hormones, adrenalectomy *et cetera* in an attempt to affect irremovable metastases. For example, some advanced carcinomata of the breast in premenopausal, or even in older, patients respond for a time to testosterone. Incidentally, the cessation of testosterone therapy may occasionally be followed by a flare-up of the metastases, whereas in other cases the opposite occurs and there is a temporary improvement. Reports have also appeared of the value of testosterone therapy for metastatic carcinomata of the ovary, thyroid gland *et cetera*, but this is still controversial. Œstrogens often cause the regression of metastatic prostatic carcinomata, but not of testicular tumours. In a significant number of cases they also cause regression of metastatic carcinomata of the female breast if the patients are well past the menopause.

ACTH and cortisone are of value in many cases of Hodgkin's disease after irradiation has lost its effect, in some cases of lymphosarcoma and leuchæmia, especially the acute forms in children and to a less extent the lymphatic variety in adults, and in advanced carcinoma of the prostate. Although not hormones, nitrogen mustard, urethane, folic acid antagonists and purine antagonists also temporarily affect conditions such as Hodgkin's disease, lymphosarcoma, the leuchæmias, and multiple myelomatosis.

Castration.

In 1941 Huggins *et alii* suggested castration for the treatment of carcinomata of the prostate, and in 1943 Huggins reported that œstrogens also produced a temporary regression of the growth and metastases in some patients. Castration has also been shown to have a beneficial effect in carcinomata of the male breast (Farrow and Adair, 1942).

Oophorectomy.

Beatson (1896) was the first to notice the palliation obtained by removing the ovaries in some advanced cases of mammary carcinoma. Since the introduction of bilateral adrenalectomy there has been a revival of the use of oophorectomy.

Adrenalectomy.

In patients with carcinoma of the prostate no longer responding to anti-androgenic therapy, bilateral adrenalectomy was carried out by Huggins and Scott (1945) in order to remove the extragonadal source of androgen; but, without cortisone, this was not a practical proposition. When cortisone became available, bilateral adrenalectomy was again tried by Huggins and Bergenstal (1952). Unfortunately the results were rather discouraging in advanced cases of carcinoma of the prostate, and "medical adrenalectomy" is now advised—that is, cortisone administration to depress the function of the adrenal cortex by a feed-back effect on the anterior pituitary lobe. This suggestion is intriguing, for it is likely that cortisone is metabolized to androgen. Huggins and Bergenstal (1952) also demonstrated that bilateral adrenalectomy often produced useful remissions in patients with metastatic mammary carcinoma. There is, however, as was concluded by Cade (1954), no accurate correlation between the response to adrenalectomy and any previous response to hormone administration or to oophorectomy. In patients with metastatic carcinoma of the breast there is more frequent relief with adrenalectomy and gonadectomy than in patients with carcinoma of the prostate, but there is not the same response to "medical adrenalectomy".

The best operative approach to the adrenal gland appears to be through the bed of the twelfth rib with the patient in the lateral position. The ovaries and one adrenal are removed at the first operation, and then, subsequently, the opposite adrenal is removed. Since the adrenal glands are friable, care must be taken to avoid incomplete removal. Any fragments allowed to remain may hypertrophy and vitiate the results of the operation. Also, a search should be made for any accessory adrenal tissue, and this should also be removed. It is really surprising that so little difficulty is associated with post-adrenalectomy maintenance except in the presence of a superadded infection or other toxic condition. In order to overcome the continued need for cortisone Galante *et alii* (1954) have suggested, after the removal of the right adrenal gland, that the left adrenal vein be anastomosed to the splenic vein so that the blood from the solitary gland will be shunted through the liver and thus the œstrogens may be prevented from reaching the general circulation. In their one case this concept was borne out, but it is an experimental procedure with little practical application. As a further attempt to study the deprivation of hormones which might be maintaining a hormone-dependent tumour, a few surgeons, such as Shimkin *et alii* (1952), have performed total hypophysectomy in a variety of patients with metastases, but the results were not conclusive. Before hypophysectomy is advocated, more work would seem to be indicated on the other means of producing atrophy of the pituitary.

Operative Treatment.

Only a few metastases are surgically removable, but, if the problem is attacked aggressively, this group may be extended to include cases which would be lost because of faint-heartedness on the part of the surgeon. Usually it is not the patients who are faint-hearted, for, if aware of the danger of metastases, they are usually willing to undergo any procedure which offers an improved chance of cure from the cancer. This point is exemplified by the acceptance of a permanent colostomy in the treatment of some carcinomata of the rectum, a procedure which patients are much more loath to accept when indicated in the treatment of serious non-malignant conditions.

"Aggressive" is defined by the Oxford Dictionary as "apt to make an unprovoked attack". However, an attack on metastases before they become troublesome, or indeed at any time, could scarcely be called unprovoked. Perhaps "belligerent" is a more appropriate word, for this is defined as "waging a regular war" and does not suggest any undeservedness. It is a sad reflection on surgical enterprise in general that it has been, and usually still is, the custom after a so-called radical removal of a cancer to await the overt signs or symptoms of metastases before again making an attack. This certainly does not suggest any aggressiveness or belligerency in attitude.

Elective Reexploration.

Reexploration of the abdomen was first described by Wangensteen in 1949. Since then, Wangensteen *et alii* (1954) have published results obtained in patients who had gastric, colic, or rectal cancers and in each of whom reexplorations were performed six months after the primary operation. Once no metastasis was found, the reexplorations were not continued. In their series of 103 cancer cases, one patient with a carcinoma of the stomach, one patient with a carcinoma of the rectum, and five patients with carcinomata of the colon had no metastasis at a final operation, even though metastases had previously been found on reexploration. For varying periods of up to fifty-one months these seven patients have remained free of metastases. This is not a high percentage, but in actual numbers it is seven who appear to have been saved.

There seems to be some doubt of the wisdom of omitting from reexploration those patients in whom, at the first operation, there was no sign of secondary invasion of lymph nodes. Wangensteen and his co-workers are now not in favour of reexploration in such circumstances, but it may be in just such a case that the odd metastasis has remained after the original operation. However, whether or not there is obvious involvement of the lymph nodes by metastases, it appears that elective reexploration should offer a better chance than a "one and only" principle in operation. If such a second operation is associated with a negligible mortality rate, it is pertinent to ask whether it is negligent not to perform elective reexplorations more often.

One objection which might be raised to the use of elective reexploration is the possibility that there would be a swing towards more incomplete primary operations when there is a second chance to deal with the cancer. On the contrary, the lessons learnt from the positions of metastases found at reexploration have emphasized the need for more extensive primary resections for certain lesions. Although the economic factor of a second or subsequent operation admittedly exists, it must be balanced against what could be the only chance of saving the patient. However, I have not had a patient object to reexploration on economic or other grounds. He or she is more concerned with any increased chance of cure.

It is not inferred that all patients who are symptom-free three to six months after operation for cancer should be submitted to exploratory operations. Rather, this is a point to be carefully considered and discussed with the patients suffering from some kinds of malignant growths, especially those of the colon, rectum and ovary, in which the prognosis is relatively good and in which, because of a likelihood of fewer metastases, reexploration should offer a greater chance of success. On the other hand, perhaps it should be just as carefully considered and discussed when the prognosis is very poor, for example, after the removal of a carcinoma of the stomach, and it is a question whether more persuasion for reexploration on the part of the surgeon may then be justified.

It is very doubtful whether the radical resection of a carcinoma of the head of the pancreas is ever indicated. Even if such a patient has withstood both the operation and the post-operative period, the five-year survival rate is

still almost nil. It is not impossible that some improvement would follow reexplorations in these cases. The classification of an excised carcinoma of the rectum as a C_2 or C_1 case (Dukes), that is, whether or not lymph nodes contain metastases up to the most proximal ligature on the mesentery, may influence the decision as to the time or desirability of reexploration. For example, classification of the case as C_2 may justify an earlier reexploration. Apart from all that, in those cases in which a second operation has been required for the eradication of a metastatic mass, and in which there is no evidence of any other metastasis, there would seem to be no doubt that, subsequently, an elective reexploration should be strongly recommended. In addition to elective reexploration of the abdominal cavity, reexploration of other likely sites of metastatic spread may be performed. When a recurrence is recognized in the perineal wound after excision of the rectum for cancer, wide excision is urgently required. However, if surgery is delayed until this stage has been reached, the position will often be found to be irretrievable, and this is one of the worst complications that can happen to the patient with a carcinoma of the rectum. Thus, reexploration of the perineal wound would seem to be advisable whenever it is suspected that the dissection has been unusually close to the growth.

The need for, and the value of, persistence in the treatment of cancer is well shown by one of my cases. This woman, aged twenty-six years, had metastases from a carcinoma of the ovary, but elective reexplorations eventually produced an apparent cure. The first operation of oophorectomy was performed on October 11, 1950, and subsequently seven reexplorations were performed. Metastases were discovered and removed during only the first four of these procedures, the last time being on October 8, 1952. Although carcinoma of the ovary occasionally behaves in an unexpected manner, there seems no doubt that her present freedom from metastases and good health are related to the treatment.

My experience with elective reexploration in other cases includes two cases of carcinoma of the stomach, one with and one without lymph node metastases in the excised specimen. No metastasis was found in either case on reexploration. In the first patient three reexplorations were performed, in the second patient one. Both these patients are alive and well although four and two years respectively have elapsed since their partial gastrectomies.

There was also a patient with carcinoma of the splenic flexure with involvement of lymph nodes. Two years after the original resection, Dr. R. Melville performed an enteroanastomosis for intestinal obstruction owing to a metastatic mass. Six weeks later this mass, together with the attached bowel and two omental metastases, was excised. After a further four months elective reexploration revealed five omental and aortic lymph node metastases, which were removed. There was no evidence of other metastatic spread. The patient is now alive and is keenly awaiting further reexploration.

Of three patients with carcinoma of the rectum, one had and two had not lymph node metastases in the excised specimen of the first operation. The former one alone showed further metastases at the time of the reexploration, and death has since occurred. The other two patients are still apparently free of metastases some two and three years later.

One patient had a primary malignant hepatoma for which a partial hepatectomy had been performed by Dr. L. Buchanan in 1948. A local recurrence and omental metastases were removed by Dr. R. J. Malcolm after a further thirty-three months and again after another eighteen months. Elective reexplorations six months later, and on three occasions since then, have revealed various metastases which were removed. Her general condition is good, and there has never been any evidence of metastases outside the abdominal cavity. She is awaiting her fifth exploratory laparotomy.

Supraradical Surgery.

Supraradical cancer surgery does not involve the performance of indiscriminate radical operations. Rather, after careful selection and consideration of the patient's general condition, the site of malignancy and the tissues involved, and the subsequent invalidity of the patient, supraradical procedures are justified in certain cases. There is no virtue in an extended radical operation performed purely because it was within the ambit of the surgeon. It is important to realize when further operative interference can only increase the misery and mutilation without hope of any palliation. On the other hand, the criteria of inoperability will often depend on the experience, ability, enterprise, and enthusiasm of the surgeon, and will vary accordingly. With certain cancers, such as those of the floor of the mouth, melanomata *et cetera*, extended radical procedures probably have a greater usefulness in the early stages of the disease than later on.

It is a fact that if metastases are not removed by the conventional so-called "radical" operations, they are usually too widely distributed to be removed by any extension of these operations. Even so, it is only by the experimental use of such procedures and a later analysis of the results that it will be possible to evaluate their usefulness. However, with certain lesions it is already apparent that the supraradical operations have little to offer. For example, although Miller and Claggett (1951) have reported survival for more than five years after pancreatico-duodenectomy for carcinoma of the head of the pancreas, this result is so rare that most surgeons have abandoned such resection for carcinoma of the pancreas. Other operations, such as Wertheim's hysterectomy, have been performed for decades, but their value and position are still controversial.

Pulmonary and Hepatic Resections.

Occasionally, after pulmonary resection it is the pathologist who asserts that the tumour is a metastasis and not a primary tumour. Similar disconcerting reports may follow the removal of a cerebral tumour. However, for a time the patient may be better off after these inadvertent resections. Apart from this, isolated cases have been reported of elective removal of a pulmonary metastasis; but this is a desperate enterprise based on the forlorn hope that there is no metastasis elsewhere.

The discovery and recognition of what is really a solitary hepatic metastasis, which may be successfully removed, are more likely some months or years after excision of the primary growth than at the time of the original operation; and several such cases have been reported. It is to be noted that a partial hepatectomy loses many of its dangers if isolation and ligation of appropriate branches of the portal vein, hepatic artery, hepatic duct and hepatic vein are performed early in the operation. In one patient in whom numerous metastases from a carcinoma of the colon were present in the right lobe of the liver with only a few in the left lobe, I ligated the right hepatic artery in the hope that this would improve the blood supply to the left lobe. His symptoms of nausea, vomiting, malaise, weakness *et cetera* all decreased after this operation, presumably owing to improved liver function. A somewhat similar relief of symptoms has been reported by Pack (1954) after a hepatic lobectomy to remove the lobe containing numerous metastases and little functioning liver tissue. In that case the relief of symptoms was attributed to the improved blood supply of the remaining lobe. It seemed possible that a similar result might be obtained simply by ligation of the branch of the hepatic artery, and this was so.

Multiple Visceral Resection.

The finding of metastatic spread of a cancer to involve a second viscus, at a stage at which operation may still offer a chance of cure, probably more often occurs with carcinoma of the rectum than with other cancers. The enterprising surgeon will have many cases in which removal of the involved posterior vaginal wall, uterus, portion of the bladder or prostate, or a loop of small bowel in addition to the rectum has been performed. The chance of cure of the malignant process will, of course, be less if such a resection is necessary, but there will be no chance if the additional viscus involved is not resected. In cases of carcinoma of the stomach, neighbouring viscera, such as the transverse colon, part of the liver *et cetera*, may require resection when they are involved; but here

even a much smaller percentage of such procedures will be only palliative. Nevertheless, the palliation obtained may be considered by the patient to be worth while.

In a patient, aged fifty years, who was suffering from a carcinoma of the right kidney, the rewards of avoiding a fatalistic attitude to metastases have been demonstrated. Some eighteen months ago a right nephrectomy was performed together with a partial left ascending colectomy; the latter was necessary owing to infiltration by an apparently solitary metastasis. Since then there has been no evidence of a recurrence, and the patient has enjoyed good health. Since venous spread was most likely in her case, elective reexploration was not advised.

Pelvic Evisceration.

In patients in whom there is an otherwise uncontrollable malignant process localized to the pelvic tissues, but without involvement of the pelvic wall, Brunschwig and Daniel (1954) have shown that occasionally a pelvic clearance may be of value as a palliative measure or even very rarely as a means of cure. The rectum, the uterus, the vagina, the bladder, the terminal portions of the ureters, and the pelvic cellular and lymphatic tissues may all be removed. The ureters are either implanted into the colon above the colostomy, or they may be brought out to the surface of the body. Alternatively, an artificial bladder may be formed from a segment of ileum or colon.

Pelvic evisceration may be required for carcinoma of the cervix after radiotherapy has failed, or less often for carcinoma of the rectum or bladder, but its most common indication is an intractable recto-vaginal or vesico-vaginal fistula following radiotherapy for carcinoma of the cervix; and such was the indication in my two cases. As Brunschwig and Daniel (1954) pointed out, the patient with an infiltrating pelvic malignant tumour *in situ* does not die in peace, and, whenever possible, it behoves us to do what we can for palliation, even if we are not optimistic enough to believe that such a patient may rarely be cured. Some patients suffering from chronic ulcerative colitis are submitted to ileostomies, which they learn to live with and to accept. Similarly, a "wet" colostomy will be accepted by patients desirous of relief from an advanced pelvic cancer.

Carcinoma of the Œsophagus.—With the exception of those in juxtaposition to the growth, the metastases of carcinomata of the œsophagus are practically always unassailable. To date, there is little place for further aggressiveness in their treatment.

Carcinoma of the Stomach.—Because of the appalling long-term results of so-called "radical" operations for certain cancers, more extensive resections have been tried in the hope of greater success. For example, for carcinoma of the stomach wider resections were recommended. The omenta, spleen, lower part of the œsophagus and associated mediastinal structures, the whole of the stomach and perhaps part of the pancreas, together with the associated lymphatic field, were resected. However, a greatly increased mortality rate is to be expected with such a procedure, and it has now been shown that its routine use would not be followed by any appreciable rise in the long-term survival rate. There has been a swing back to more limited resections, except when the growth is placed so high up in the stomach that it would not be removed by a partial gastrectomy. Despite this, the long-term survival rates after operations for cancer of the stomach have recently shown a definite improvement. This must be owing either to a better selection of cases for operation, or to earlier diagnosis, better anæsthesia and resuscitation, chemotherapy *et cetera*. Nevertheless, reported results such as 10% five-year survivals in patients with proven lymph node metastases at the time of operation, now provide the chance to demonstrate the positions of these metastases in a significant number of cases and to base future operations on these results. However, it is to be expected that such a study on patients with carcinoma of the stomach, who have apparently been cured, will show that the metastases were very limited in number and that they were all close to the primary growth. In other words, it is considered that supraradical operations have little to offer for the routine treatment of cancer of the stomach and its metastases. For a patient with a carcinoma of the stomach, palliation may be required because of symptoms produced by the primary growth; and this is best obtained with a gastric resection. Also, it has been suggested (State *et alii*, 1950) that there is a substantial prolongation of the survival period when such resection has been undertaken in the presence of hepatic metastases. However, other palliative operations are rarely required in the management of metastases from carcinoma of the stomach.

Carcinoma of the Rectum.—As with carcinoma of the stomach, similar attempts have been made to extend the operations for carcinoma of the rectum. Excision of the lymphatic field up to the origin of the inferior mesenteric artery has recently been revived by many surgeons. In addition, Deddish (1950) and others have proposed wider resections of the pelvic cellular tissues. These extensions of the abdomino-perineal operation are certainly determined attempts to deal with metastases; but, as with other supraradical or extended procedures, there is some doubt whether such extensions will result in more long-term survivors. By their use it is inferred that the only affected lymph nodes which have remained after the routine operation would be included by a slightly wider excision of the lymphatic field, and that this would save more patients than would be lost because of an increased operative mortality rate. It seems fanciful to hope that the spread of the metastases would have gone just so far and no further in more than the exceptional case.

Carcinoma of Vulva and Anus.—The need for more extensive lymph gland dissections has been emphasized and practised by Stanley Way for carcinoma of the vulva. His procedure, which includes dissection of the inguinal and external iliac lymph glands on each side, is widely accepted. Similar operations are not usually performed for carcinoma of the anus, but this is a lesion with a relatively bad prognosis, and more aggressive attempts to deal with its metastases would seem to be indicated.

Carcinoma of the Breast.—Because of the poor long-term results of treatment of carcinoma of the breast, various attempts have been made to extend the scope of routine radical mastectomy to include metastases which may be present in the cervical and mediastinal regions. For example, Urban (1952) includes the internal mammary chain and anterior mediastinal structures with the breast and axillary tissues and, like Handley and Thackray (1949), he has found that the internal mammary lymph nodes may occasionally be involved in the absence of axillary metastases. However, while removal and examination of these nodes may be of help in estimating the prognosis, it is doubtful whether such extensions of the operation to the mediastinum will alter the long-term survival rate. For the present these extended operations remain experimental, and there is no place for indiscriminate supraradical mastectomy. Attempts are again being made to attack the problem of carcinoma of the breast by more restricted procedures. However, it is probable that McWhirter's (1948) provisional figures for simple mastectomy followed by radiotherapy will be significantly affected with the passage of time, when any surviving cancer cells are no longer restrained by the irradiation.

Malignant Melanoma.—Malignant melanomata are rarely found before puberty; but in one of my patients, aged ten years, there was no evidence of precocious puberty. Also, malignant melanomata are said not to develop in castrated individuals (Shimkin *et alii*, 1952); and the growth of the tumour is often accelerated by puberty or pregnancy. While it appears that these growths are under the control of hormones, there is no evidence, once a malignant melanoma has developed, that it is affected by androgens, œstrogens, cortisone, or other hormones, or by castration, by oophorectomy or by bilateral adrenalectomy. The futility of treating metastases from a malignant melanoma by operations such as forequarter and hindquarter amputation is now realized. Wherever possible, regional lymph node dissections appear to be advisable at the time of excision of primary malignant melanomata, but the value of elective reexplorations of the lymph nodes at a later stage is in doubt.

Summary.

The present position of the diagnosis and treatment of metastases is discussed. It is believed that, while the future treatment of metastases lies more with hormonal and other methods, surgical attack holds pride of place at present, and in most cases should be employed as far as conditions allow. In addition to the use of surgery after metastases have been detected clinically, there is also a place for elective reexploration to search for and to deal with metastases before they are otherwise detectable. In the absence of irremovable metastases at the primary operation, elective reexploration of the abdomen should be considered within six months in cases of carcinoma of the colon, ovary, liver, pancreas, and rectum, and slightly earlier in cases of carcinoma of the stomach. In all cases in which a second operation has already been required for the eradication of a metastatic mass, and where there is no evidence of any other metastasis, an elective reexploration should be strongly recommended. The place for supraradical surgery is in the early stages of certain cancers rather than in the subterminal stages of the disease. However, it is not always futile to perform a massive resection of metastases; and occasionally even supraradical surgery is indicated for palliation. A case is described in which symptoms of hepatic insufficiency were temporarily relieved by ligation of the branch of the hepatic artery to that particular lobe destroyed by the metastases. It is considered that the relief was due to the resulting increased blood supply of the lobe which contained most of the functioning hepatic tissue.

It is concluded that more study is indicated of: (i) the effects on cancer cells of substances which stimulate the growth or metabolism of normal cells, (ii) the reasons why some malignant growths do not metastasize, (iii) the use in "hormone-dependent" cancers of hormone therapy and of hormone exclusion operations, especially hypophysectomy produced by both surgical and medical means, (iv) the place of elective reexploration in the management of intraabdominal and other cancers, and (v) the positions of metastases in specimens removed from patients apparently cured by surgery. Such a study would give a basis for determining the extent of operations to be employed for various cancers.

Acknowledgement.

In a "Current Comment" in THE MEDICAL JOURNAL OF AUSTRALIA of February 13, 1954, it was stated that adrenalectomy calls for considerable skill on the part of the anæsthetist. It certainly does. Most of the anæsthetics for adrenalectomy in Sydney have been given by Dr. Gwenifer Bernard, and I would like to thank her for her care of and interest both in these and in other patients of mine, especially those in poor condition and in whom extensive procedures have been required.

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PELVI-URETERIC OBSTRUCTION.

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Sydney.

THE destruction of a kidney over a period of years by obstruction to the pelvi-ureteric outlet is not an uncommon occurrence. The adoption of conservative medical measures of treatment leads only too often to nephrectomy. This, of course, may be unavoidable; but the price is paid by the loss of functional renal tissue. When the kidney is pyonephrotic or has lost all function, then removal is inevitable. Short of this, hope need not be abandoned, for experience shows that the elimination of obstruction can permit the return of an amazing degree of function (Case II), and, surprisingly enough, even anatomical restoration (Case I).

Onset.

Persons suffering from this condition may present at any age, but the majority enter the diagnostic forum in early adult life and under one of two guises: firstly, with a long history of recurrent unilateral "pyelitis" (for the disease rarely involves both sides); or secondly, with a short history of loin pain. The former is easy to understand, because the basic pathological condition is of long standing and often congenital. The latter defies our knowledge, for it is not uncommon to discover in such cases a gross degree of hydronephrotic atrophy of the kidney. Any obstruction found is always obviously of long standing; yet previous pain or evidence of infection has often been absent.

What happens to precipitate the onset of pain so suddenly? The answer is quite unknown. When infection is present, this can be accepted as a cause; but just as often no evidence of such a state can be produced.

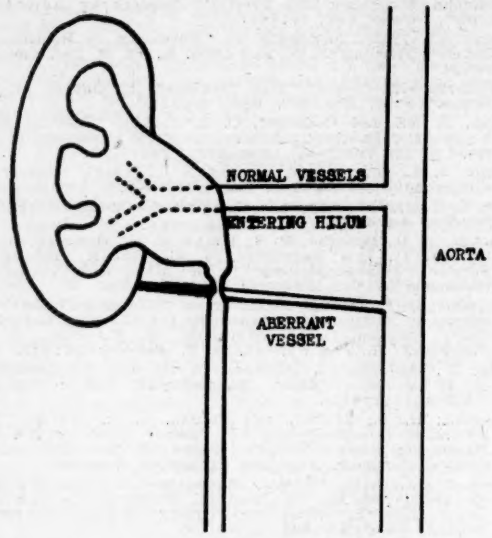

FIGURE I.
Condition exposed at operation. Aberrant vessel to lower pole combined with stricture of the ureter just below the pelvi-ureteric junction.

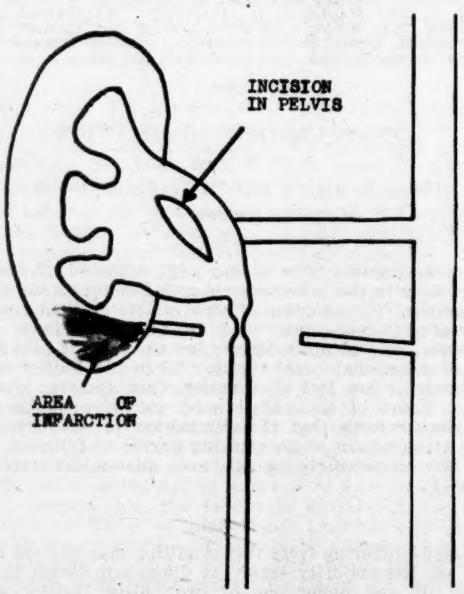

FIGURE II.
Vessel ligated and removed. A variable area of infarction appears. This is rarely extensive. The dilated pelvis is opened by an incision in its long axis.

Ætiology.

Hydronephrosis due to a mobile calculus in the pelvi-calyceal system is excluded from the discussion, as also are the effects of extrinsic pressure upon the pelvi-ureteric area of perirenal post-infective fibrosis. The recurrent semi-obstructions of the hypermobile kidney are also not considered here. Two factors only play a part in the production of the hydronephrosis under discussion. These are the presence of aberrant vessels—arterial, venous or

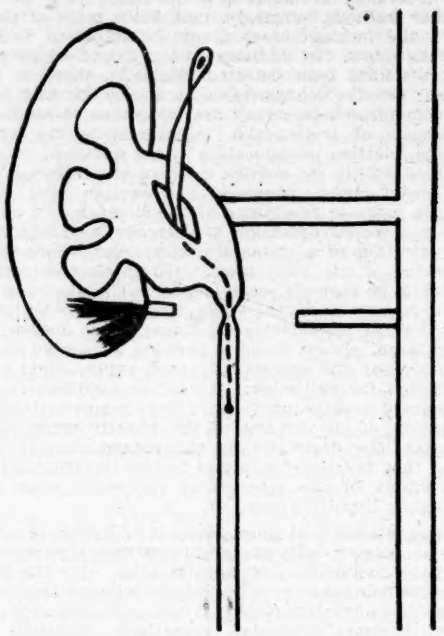

FIGURE III.
A probe is passed down into the ureter, dilating the stricture.

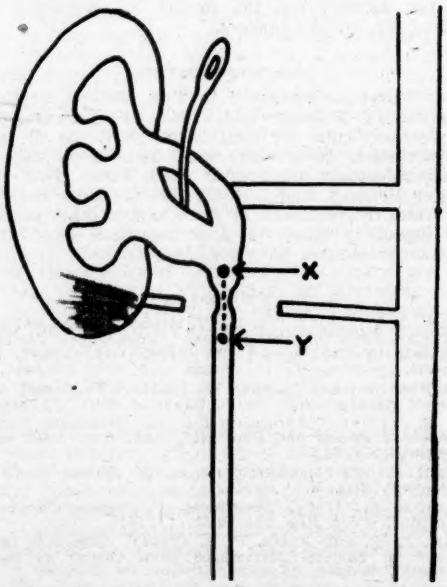

FIGURE IV.
An incision is made from X to Y in the long axis of the ureter. It passes through all coats. X and Y are beyond the limits of the stricture.

both—and a true intrinsic stricture of that portion of the ureter adjacent to the pelvi-ureteric junction. It is, of course, possible to postulate a congenital pin-point orifice between the renal pelvis and the ureter in the absence of any other pathological factor, but this seems to be quite uncommon.

It has been demonstrated that aberrant vessels and intrinsic stricture do coexist, and because of this no surgeon cuts such vessels without also opening the renal pelvis and proving the absence of a ureteric stricture. But even the finding of one gives no final decision on the question of ætiology, for after all, inflammation is the uniform response of all tissue to any injury, and the life-

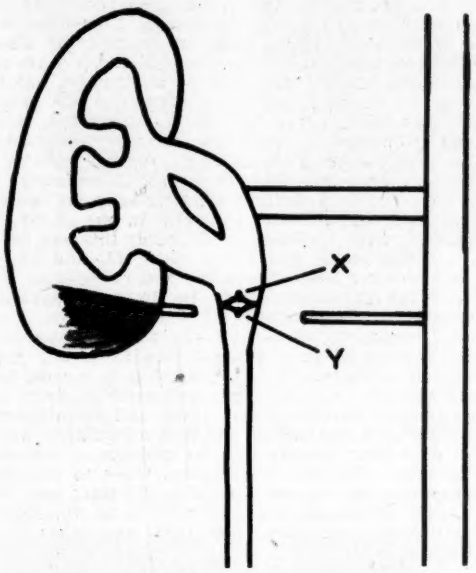

FIGURE V.

The points X and Y are drawn together by a catgut suture. This serves to convert the longitudinal axis of the incision XY into a transverse one. The result is a wide opening between the pelvis and the ureter. Several interrupted catgut sutures close the incision.

long rubbing of the mobile pelvi-ureteric area upon an aberrant vessel is just another injury. The stricture may well be the horrible child of its evil parent.

Just as often a stricture in this area of the ureter is present when no other factor can be shown to be operating or can be deduced from a careful history. It is, of course, possible that "subclinical" local infections, of which the patient and parents are ignorant, may be the cause. This could be so, but is certainly "not proven".

It must be accepted by the clinician that either or both factors may operate in any individual case. The point is that both are remediable by operation even at a late stage. It can also be said that the advent of the antibiotic drugs have so tamed the infective organisms that what were once relative indications for plastic procedures have now become absolute.

Diagnosis.

The existence of the condition is demonstrated by intravenous and retrograde pyelography. Urine culture establishes the presence of infection, and sensitivity tests indicate the appropriate preliminary antibiotic measures. The lack of concentration of dye (as in Case II) is not cause for despair. Nephrectomy is not compulsory because of this finding.

Vascular Anatomy.

It is wise at this juncture to decide upon a definition for an aberrant vessel. An artery or vein which is truly aberrant must enter the kidney parenchyma other than by the hilum of the organ. The point of origin of such a vessel from the parent trunk is of little moment. However, this much can be said—the closer the point of origin of the abnormally placed vessel to the renal hilum, the less the pathological significance. Furthermore, it is rarely found that aberrant vessels to the upper pole cause trouble.

"False" Aberrant Vessel.

A "false" aberrant vessel may be found at operation. There is invariably considerable dilatation of the renal pelvis, and the true renal veins and arteries are stretched out over it. The ureter is hooked up over one or more of the vessels, which enter the hilum in a normal fashion. It is easy to imagine this to be the cause of obstruction, and it is, in fact, at the time of operation. However, the vessel concerned is not aberrant. It may come as a branch from the parent vessel or even from the main axial vessels,

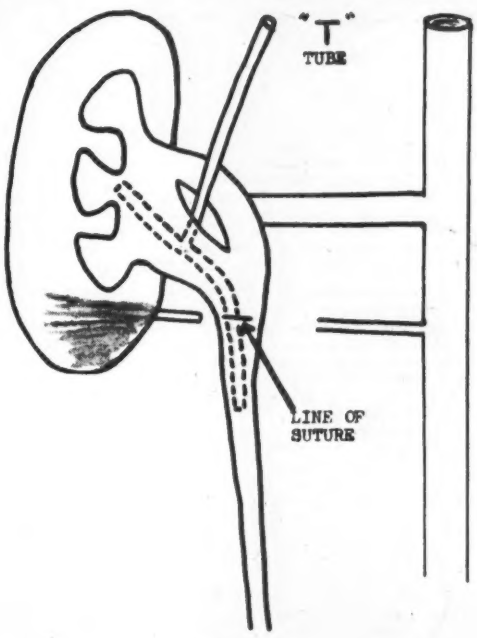

FIGURE VI.

A "T" tube is placed so that one arm splints the pelvi-ureteric area and the other drains the urine from the pelvis. The length of the arms is adjusted to suit the individual case. The incision in the pelvis proper is closed around the "T" tube. A wide-bore rubber tube is used to drain the perirenal space in the usual manner after renal surgery. The "T" tubes used for drainage of the common bile duct are adequate for the purpose of this operation.

but it enters the kidney parenchyma by the hilum. It is the expanding renal pelvis pushing upon vessels that distorts a normal anatomical relationship and produces a secondary obstruction well on in the course of the disease. Ligation only of such a vessel is futile.

Treatment.

The treatment of this condition is by operation. The choice lies between nephrectomy and reparative procedures. The older the patient, the less the function of the kidney and the greater the degree of infection, the more is nephrectomy indicated. This is so if the other kidney is normal; otherwise, if possible, more conservative measures are adopted.

The antibiotic drugs have permitted plastic operation in cases in which infection existed which were formerly foredoomed to failure, and it is astonishing what a degree of functional recovery may take place in a gravely damaged kidney after successful operation (Case II).

Technique.

A simple and reliable technique, in which both an aberrant vessel and a ureteric stricture are found, is illustrated in the diagrams (Figures I to VI).

After Care.

The loin drainage tube is removed by the fourth day and the "T" tube at the end of the third week. There is no difficulty in its removal, and surprisingly enough the sinus closes within a few hours.

Serial urine cultures are essential, and antibiotics are used if sensitive organisms appear. Infection, if not pre-existing, is quite rare. The patient becomes ambulant at the end of the second week, with the tube draining into a small flat bottle strapped to the thigh.

Reports of Cases.

CASE I.—In this instance the patient, a woman, aged twenty-eight years, complained of pain in her right side. This was becoming more severe and constant. Symptoms had been present for three years. No infective element was demonstrated. At operation a small aberrant vessel arising from the aorta was found, passing into the lower pole of the kidney. There was also a definite stricture of the ureter at the site where the vessel and ureter were in contact. The radiographs (Figures VII and VIII) show the striking return to anatomical normality in this case. This patient is still under observation and is well, after two years.

CASE II.—In this instance the patient, a boy, aged fifteen years, complained of pain in the right side "for years". There had been episodes of acute cramp in the side associated with feverishness. The patient was referred for urological examination because he was always ill. The intravenous urogram showed no excretion of dye on the right side. There was no evidence of pus cells or organisms in the urine. Attempts at culture gave negative results. Retrograde pyelographic studies were not made in this case at the express wish of the parents. At operation no aberrant vessel was found, but there was a pronounced stricture of the ureter almost half an inch long just distal to the pelvi-ureteric junction. The orifice, as seen from inside the renal pelvis, was normal in size. The pelvis was much dilated and the renal parenchyma considerably reduced. It was thought that nephrectomy could well be indicated, but the conservative operation was performed.

The follow-up intravenous urograms two months later showed a return of function to the degree of good concentration of the injected dye. This state has persisted, although there has been no evidence of restoration to anatomical normality. The boy became free of all symptoms and has since played first-grade Rugby for his school. The degree of functional response and the symptomatic cure in this case were gratifying.

Conclusion.

The possibility of the restoration of the kidney to normal anatomical structure or to a degree of worthwhile function can be demonstrated. The technical procedure is not of an arduous nature, and the discomfort to the patient is minimal.

Legends to Illustrations.

FIGURE VII.—Case I: Showing the classical picture of pelvi-ureteric obstruction.

FIGURE VIII.—Case I: After operation, showing a return to normal structure with a good funnel-shaped pelvi-ureteric opening.

FIGURE IX.—Case II: No evidence of dye secretion on the right side.

FIGURE X.—Case II: After operation. Secretion of dye is occurring on the right side.

OIL GRANULOMA OF THE LUNG (LIPOID PNEUMONIA).[1]

By M. P. SUSMAN,
Sydney.

SINCE LAUGHLEN (1925) first drew attention to the danger of inhaling oil, over 400 cases of lipoid pneumonia have been reported. Jampolis, McDonald and Claggett (1953), in reporting a case from the Mayo Clinic, reviewed the history and other features of this condition and gave an extensive bibliography. They also described a method for detecting the type of oil by chemical analysis of the lesion. Fat in the lung may be endogenous or exogenous. Endogenous fat may be due to fat emboli, to lipoid histocytic disease, or to fat that has escaped from cells as the result of infection. Exogenous fat reaches the tracheobronchial tree directly. The commonest offenders are nasal oil drops and mineral oil taken as a laxative; but it is salutary to remember that iodized oil used for bronchography may be the causative agent. Symptoms may be few and unimpressive; generally they are those common to most lung lesions, such as cough, sputum, pain or discomfort and dyspnœa. It is important to remember that hæmoptysis may be a leading symptom, as in the two cases reported here. A confusing symptom in one of my cases was paresis of the ipsilateral vocal cord; this was hard to explain, as the lesion was on the right side and was confined to the lower lobe. The nerve was not seen at operation and the explanation may lie in some anatomical abnormality, or the paralysis may have been due to some unrelated condition. Sometimes the patient comes to the surgeon because of an undiagnosed radiologically opaque area, which varies from a diffuse mottling to a dense homogeneous shadow. A history of using nasal oil drops or of imbibing liquid paraffin is important, and examination of the sputum after the patient has been on a fatless diet for several days may disclose free fat globules or vacuolated macrophages. For this examination, three to five whole-day specimens are stained with Wright's stain and Sudan IV. Lipoid pneumonia has especially to be differentiated from neoplasms and from tuberculosis and other chronic infections.

Treatment.

There is no specific medical treatment, but chemotherapy is useful for infective complications. Because of this risk of infection, removal of the lesion should be advised if there are no contraindications. Often the operation is primarily exploratory, and the diagnosis may be established only on microscopic examination. Operation should be as conservative as possible.

Reports of Cases.

CASE I.—A man, aged sixty-four years, had had a cough for five years. About six months prior to examination he had had an acute respiratory infection from which he did not ever fully recover, in that he lost weight and energy, the cough had become more troublesome and the sputum was now occasionally blood-stained. There were physical and radiological signs of partial consolidation of the lower lobe of the right lung (Figure I). No abnormality was found on bronchoscopic examination except paresis of the right vocal cord, and no cancer cells or special organisms were found in the sputum. (It was not until after operation that a history of taking petroleum jelly for indigestion was obtained.) At operation the lower lobe of the right lung was found to be almost solid, and some soft enlarged hilar lymph glands were noted. There was a general impression that the lesion was inflammatory rather than neoplastic, and for that reason no frozen section was made. The lobe was removed (Figure II), and microscopic examination showed it to be the seat of lipoid pneumonia (Figures III and IV). Two years later the patient was working and well, and there were no clinical or radiological abnormalities.

CASE II.—A man, aged fifty years, had suffered from frequent severe "colds" for the last nine years. He had become progressively dyspnœic in the last eighteen months, and for

[1] Read at the third International Congress on Diseases of the Chest at Barcelona, 1954.

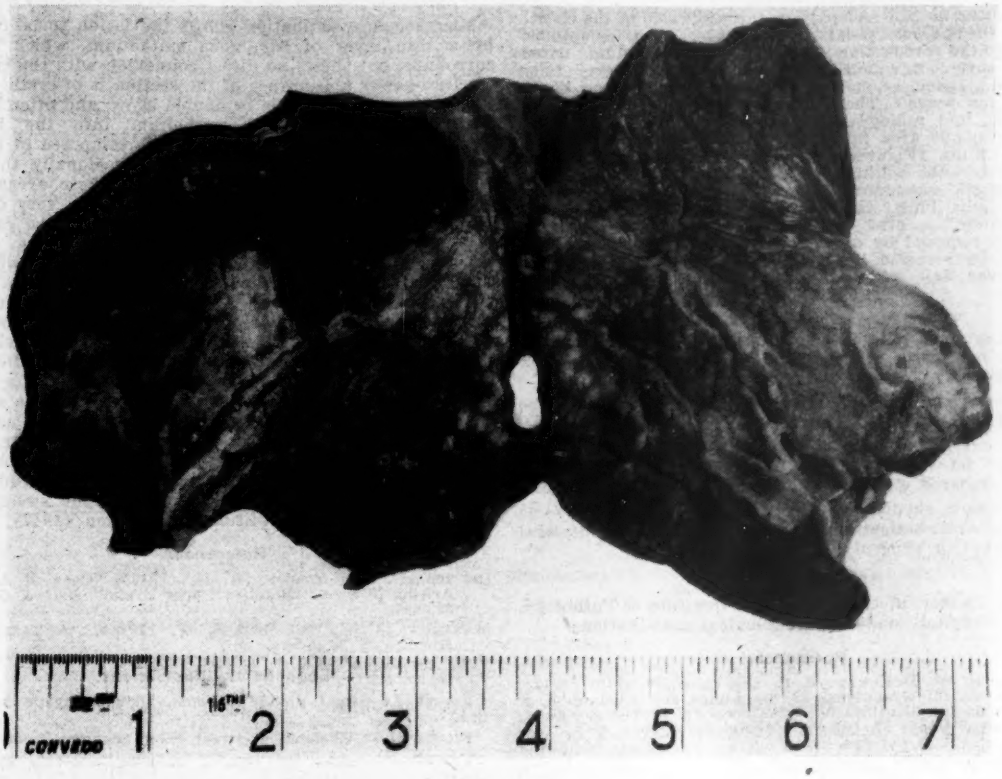

FIGURE II.
The lower lobe of the right lung has been divided longitudinally and the two halves have been folded outwards.

FIGURE III.
Low-power section stained with hæmatoxylin and Sudan IV. There are fibrosis and infiltration with lymphocytes, plasma cells and polymorphonuclear cells. Phagocytes laden with Sudanophile droplets are present in alveoli.

FIGURE VII.
This is the middle lobe of the right lung; most of it is solid.

three months he had had pain in the right side of his chest; recently he had lost weight. The more serious symptoms, including the production of blood-stained sputum, dated from a severe infection from which he had never fully recovered. Later, he stated that he had been taking liquid paraffin for years. There were physical signs of general bronchitis, and radiographic examination revealed a dense uniform opaque area in the position of the middle lobe of the right lung (Figures V and VI). Nothing remarkable was found in the sputum and no abnormality was found at bronchoscopic examination. At operation the middle lobe of the right lung was solid; the other lobes and the mediastinum appeared normal. The middle lobe (Figure VII) was removed by dissection technique, and microscopic examination revealed lipoid pneumonia. A year later the patient was well, with no abnormal clinical or radiological findings.

Summary.

Oil granuloma (lipoid pneumonia) is reviewed briefly, and two new cases are recorded. Resection of the lesion was followed by complete recovery.

A history of inhaling or drinking oily substances may be obtained, often only after direct questioning of the patient.

When oil granuloma is suspected a search should be made for fat in the sputum. The findings in a frozen section prepared during a thoracotomy may be diagnostic.

The lesion should be removed unless there is some obvious contraindication, such as diffuse or bilateral involvement or general ill health.

Acknowledgement.

Dr. A. Palmer, of the Kanematsu Institute of Pathology, Sydney Hospital, made the histological examinations.

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Legends to Illustrations.

FIGURE I.—The opaque area to the right of the heart shadow represents the solid area in the lower lobe of the right lung.

FIGURE IV.—High-power section stained with hæmatoxylin and Sudan IV, showing phagocytes laden with Sudanophile droplets in an alveolus.

FIGURE V.—The opaque middle lobe of the right lung in the postero-anterior projection.

FIGURE VI.—Lateral view of the solid middle lobe of the right lung.

Reports of Cases.

ADENOMA OF THE EYELID GLANDS OF MOLL.

By C. E. MARSHALL,
Sydney.

THE glands of Moll in the eyelid are specialized sweat glands. They differ from sweat glands in other situations, in that the terminal portion is not coiled into a ball but is more or less straight. The epithelium of this terminal portion consists of an indistinct, outer myoepithelial layer, adjacent to the basement membrane, and an inner layer of pyramidal cells of apocrine type. The lumen is often affected by cystic dilatation with consequent flattening of the lining cells, and the adenoma also displays this cystic tendency. The ducts, which usually open into the follicles of the eyelashes, are composed of two distinct cell layers. The nature of the secretion is unknown (Maximow and Bloom, 1942).

Clinical Record.

The patient was a married woman, aged twenty-six years. She had noticed a small nodule on the upper eyelid which had been present for three months. It was described in the history as a "granulation tissue mass", and it measured 0·3 centimetre in maximum extent.

Microscopic examination shows the lesion to be covered by a thin layer of squamous epithelium, which closely surrounds, but shows no direct connexion with the tumour. The low-power appearance of the section is of cystic spaces lined by cells in a single or double layer, and often having papillary projections of epithelium into the lumina (Figure I). Elsewhere, the tumour is composed of tubules which may show branching. Very occasionally the cells tend to form compact whorls, and in these areas intercellular bridges can be seen. The stroma is very delicate and consists of fine connective tissue trabeculæ carrying minute capillary vessels. The epithelial lining of the tubules is composed of cuboidal, slightly eosinophilic cells, having a large rounded nucleus with sometimes a single central nucleolus (Figure II). In some places the cells are more pyramidal and much more eosinophilic and form a single layer lining a cystic space. These cells are "apocrine-like", and are reminiscent of the cells often seen as a metaplastic change in the ductules of the breast (Figure III). Elsewhere, some of the cells have a slightly foamy appearance suggestive of sebaceous metaplasia and some are vacuolated, but no mucin has been demonstrated within the latter. The tumour is considered to be a benign adenoma of modified sweat ducts. Its structure is very similar to the photomicrographs of the sweat gland adenoma described by Clifton and Gordon (1947).

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Legends to Illustrations.

FIGURE I.—Sweat gland adenoma, showing cystic structure (× 81).

FIGURE II.—Cell detail of sweat gland adenoma (× 450).

FIGURE III.—Area of tumour showing apocrine cystology (× 450).

A CASE OF CONGENITAL SPHEROCYTIC ANÆMIA WITH AN AREGENERATIVE CRISIS.

By D. WHITE.

From the Department of Pathology, Royal Alexandra Hospital for Children, Sydney.

SEVERAL authors (Owren, Damashek, *et alii*) have now described the hæmatological changes occurring in congenital spherocytic anæmia during the period of a crisis. In a previous article, Margolis reported three examples of aplastic crises as they occurred in three patients suffering from congenital spherocytic anæmia. In only one of these cases was the opportunity afforded to examine the marrow in the aplastic phase, and in this case only in the late stages of this phase. A further case, in which repeated examination of the bone marrow revealed the aplastic phase as well as the rapid changes which occur during recovery from this phase, has now been investigated.

Clinical Record.

The patient was a boy, aged four years, who had a family history which was very suggestive of a congenital hæmolytic disorder. His father was known to have suffered from congenital hæmolytic anæmia, while two of his paternal uncles and one cousin had been subjected to splenectomy for this same reason. The diagnosis, in the present case, was supported by the laboratory investigations which included the finding of microspherocytes and an increased osmotic fragility of the red cells to saline both before and after incubation. Splenectomy was not performed at this time, and on three occasions during the subsequent three years the boy was admitted to hospital for blood transfusions.

On August 18, 1953, he was admitted to hospital again because of pyrexia and a severe upper respiratory tract

infection which had been present for eight days. Coincidently with this infection he had been noticed to become increasingly pale. At the time of his admission to hospital he was obviously pale; no jaundice was evident, and the liver and spleen were not palpably enlarged. A blood count at this time showed the following figures: the hæmoglobin value was 4·5 grammes *per centum*, the erythrocytes numbered 1,700,000 per cubic millimetre, the colour index was 1·11 and reticulocytes were absent; the leucocytes numbered 6000 per cubic millimetre, 60% being polymorphonuclear cells, 28% lymphocytes and 12% monocytes. Thrombocytes numbered 400,000 per cubic millimetre. On examination of a blood smear, a large proportion of the red cells were seen to be spherocytic.

After the patient's admission to hospital, serial investigations were performed and gave the results shown in Table I.

TABLE I.

Date.	Hæmoglobin Value. (Grammes *per Centum*.)	Red Cells per Cubic Millimetre.	Proportion of Reticulocytes.	Remarks.
18/8/53	4·5	1,700,000	Nil	Coombes test result negative.
19/8/53	8·2	2,600,000	Nil	Transfusion of 500 millilitres after admission to hospital.
20/8/53	8·8	2,600,000	Less than 0·5%	Urinary urobilinogen content greatly increased.
21/8/53	8·5	2,430,000	Less than 0·5%	Urinary urobilinogen content greatly increased.
24/8/53	9·5	2,940,000	7·0%	No urinary urobilinogen; nucleated red cells 2000 per cubic millimetre.
26/8/53	10·5	3,330,000	20·0%	No urinary urobilinogen; nucleated red cells, very occasional.
28/8/53	10·5	3,500,000	16·0%	—

Two iliac crest marrow punctures were performed, one on the patient's admission to hospital and a second one sixty hours afterwards, with the results shown in Table II.

The patient was discharged from hospital twelve days after his admission.

On September 18, 1953, he was readmitted to hospital because of pyrexia and a mild respiratory tract infection, which had been present for five days. Once again, coincidently with this infection, he had become increasingly pale. At the time of his admission to hospital he was a pale child with a slight icteric tinge, and with no palpable enlargement of his liver and spleen. A blood count on his admission to hospital gave the following figures: the hæmoglobin value was 8·0 grammes *per centum*, the erythrocytes numbered 3,000,000 and the colour index was 0·85; the leucocytes numbered 18,000 per cubic millimetre, 77% being polymorphonuclear cells, 13% lymphocytes, 3% monocytes and 7% eosinophile cells; thrombocytes were numerous. Examination of a blood smear revealed slight anisocytosis, with many polychromatic cells and a number of spherocytes.

The patient was given a blood transfusion, and it was decided to perform a splenectomy at a future date. This was done on February 12, 1954. The spleen was enlarged, weighing 255 grammes, and examination of it revealed the characteristic findings seen in congenital hæmolytic anæmia. After an uneventful post-operative course, the patient was discharged from hospital.

On July 20, 1954, he was readmitted to hospital because of drowsiness, vomiting and loose bowel motions. On examination, he was found to have slight neck stiffness and slight spine stiffness. Lumbar puncture was performed, and *Hæmophilus influenzæ* organisms were isolated from the turbid fluid. He rapidly responded to treatment with antibiotics and made an uneventful recovery. A blood count at that time gave the following information: the hæmoglobin value was 14·0 grammes *per centum*; the leucocytes numbered 29,000 per cubic millimetre, 71% being polymorphonuclear cells, 20% lymphocytes, 5% monocytes and 3% metamyelocytes; thrombocytes were numerous. On examination of a blood smear, the red cells appeared normal to microcytic in size, many cells being microspherocytes; their shape and colour were normal.

Since his discharge from hospital the patient has regularly attended the follow-up clinic and has remained well.

TABLE II.

Cells.	Date.	
	18/8/53.[1]	21/8/53.[2]
Myeloblasts ..	0·5%	1·0%
Promyelocytes	1·0%	1·5%
Myelocytes ..	19·0%	18·5%
Metamyelocytes	31·0%	21·5%
Polymorphs ..	32·0%	30·0%
Lymphocytes ..	13·0%	12·0%
Monocytes ..	1·5%	0·5%
Degenerate ..	2·0%	Nil
Nucleated red cells	Less than 0·5%	18% { 4·0% Proerythroblasts. 10·0% Early normoblasts. 2·0% Intermediate normoblasts. 2·0% Late normoblasts.
Megakaryocytes	Present in normal numbers.	Present in normal numbers.

[1] See Figure I.
[2] See Figure II.

Discussion.

As in the cases previously reported, there occurred a sudden cessation of erythropoiesis, apparently precipitated by an external disturbance in the form of a respiratory tract infection, resulting in an almost complete absence of red-cell precursors from the bone marrow, and an accompanying absence of reticulocytes in the peripheral blood as well as an absence of urobilinogen from the urine. In this case, the leucopenia and thrombocytopenia reported by others (Owren and Damashek) were not a feature of the aplastic phase.

The occurrence on September 18, 1953, of the symptoms of pyrexia, a mild respiratory tract infection, and increased pallor is of interest, particularly when they were accompanied by the finding of "numerous polychromatic" cells (reticulocytes). This episode, although not fully investigated, may well have been a true hæmolytic crisis. Castle has recently discussed this interrelation between aplastic and hæmolytic crises in congenital spherocytic anæmia.

After the splenectomy, the onset of a severe infection (meningitis), while not of the "virus" type normally associated with the onset of an aplastic crisis, produced no alteration in the blood picture.

Summary.

A case of congenital spherocytic anæmia is described, and the findings in the bone marrow during a crisis are reported.

Acknowledgements.

My thanks are due to Dr. W. MacCallum for permission to publish this case, and to Dr. R. D. K. Reye for his helpful criticisms. The photomicrographs were taken in the Photographic Section of the Department of Pathology, Royal Alexandra Hospital for Children.

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MARGOLIS, J. (1953), "Aplastic Crises in Congenital Hæmolytic Icterus (Acholuric Jaundice): A Description of Three Cases", M. J. AUSTRALIA, 2:100.

OWEN, P. (1948), "Congenital Hæmolytic Jaundice: The Pathogenesis of the Hæmolytic Crisis", *Blood*, 3:231.

Legends to Illustrations.

FIGURE I.—Bone marrow (August 18, 1953), showing complete absence of the red-cell precursors; white-cell precursors in normal numbers (×200).

FIGURE II.—Bone marrow (August 21, 1953), showing early red-cell precursors, with normal white-cell precursors (×400).

BILATERAL CARCINOMA OF THE FALLOPIAN TUBE.

By C. E. MARSHALL,
Sydney.

PRIMARY CARCINOMA of the Fallopian tube is both very rare and very malignant; Anderson states that only about 500 cases have been described in the literature, but increasing numbers have been reported during the past twenty-five years. Because of its rarity a pre-operative diagnosis is seldom made, although it is a tumour which can fairly readily be palpated, and one in which the pain accompanying it is consistently described as "colicky" or "crampy". This pain is situated in the lower quadrant of the affected side and radiates to the back; and this is one of the few instances in which pain is a diagnostic accompaniment of early carcinoma.

Although no age is exempt, it is a tumour which occurs most frequently around or after the menopause, when the first and commonest sign of the lesion is a blood-stained vaginal discharge. The discharge is said to have the sero-sanguinous appearance of "orangeade". Israel (1954) was able to make the correct pre-operative diagnosis in two cases because of the presence of this feature.

In one-third of cases the tumours are bilateral, and this is an interesting occurrence in view of the fact that several ovarian neoplasms may also be bilateral. Metastasis to the ovary and uterus takes place via the lymphatics and to the abdominal cavity by means of peritoneal "spill". Curettage usually fails to reveal the diagnosis, but examination of the sero-sanguinous discharge by the Papanicolaou technique may disclose the presence of malignant cells.

Ayre (1945) states that multiple pelvic lesions such as chronic salpingitis, ovarian cystomata and uterine fibroid tumours have been found in most cases; but these conditions are so common that their occurrence can have no direct causal relationship to the carcinoma. However, tuberculosis seems to be more frequently coincident than one would expect, and may be of importance in stimulating the formation of benign papillomata which subsequently become malignant (Fullerton, 1940).

The tumour tends to grow inwards towards the lumen of the tube in a papillary fashion so that the tube becomes greatly distended. The wall of the tube is rarely invaded, at least until very late, so that the peritoneal surface is smooth and free from growth. Nevertheless, the prognosis is very poor (Goldberg, 1954).

Various histological types have been described, but basically the tumour is a papillary carcinoma, the structure of which usually resembles that of the Fallopian tube in an exaggerated form. Any variation of this arrangement, such as the pseudoglandular or alveolar patterns sometimes described, is due to the poor differentiation of the lesion, and not to the presence of a different variety. For example, adenocarcinoma does not occur because the normal Fallopian tube contains no glands. Well differentiated tumours have been described in which the growth has been confused with tuberculosis, and Fullerton (1940) states that he has seen several instances of tuberculosis reported as primary carcinoma. In the case to be described, no such confusion is possible because of the numerous mitotic figures present and the atypical cell types seen.

The accepted treatment is total hysterectomy with bilateral salpingo-oophorectomy, with or without post-operative pelvic irradiation.

Clinical Record.

The patient was a white woman, aged fifty-three years, who said that her menstrual history had been quite regular until two years previously, when the periods started to become less frequent and more profuse. The final menstrual period had occurred four months prior to her admission to hospital, and since then she had noticed a continuous, profuse, watery discharge. About three months prior to her admission to hospital she had first noticed enlargement of her abdomen, and she was able to feel a tumour therein. At times she suffered from sharp, brief cramps in the abdomen. At the age of nineteen years she had pleurisy, but there was no history of tuberculosis or salpingitis. She was married, but had had no children or miscarriages.

On examination of the patient, a rounded, firm tumour was felt, which extended from the pubis to the umbilicus. It was tender on palpation, and the pre-operative diagnosis of a uterine fibroid tumour was made.

At operation, a grapefruit-sized cyst was found which involved most of the left Fallopian tube and an orange-sized cyst which involved the right tube. The tubes were found to be filled with creamy, friable, partly necrotic papillary neoplasm. The growth was centripetal, and the outer surfaces of the tubes were free from tumour (Figure I). The ovaries and uterus appeared normal.

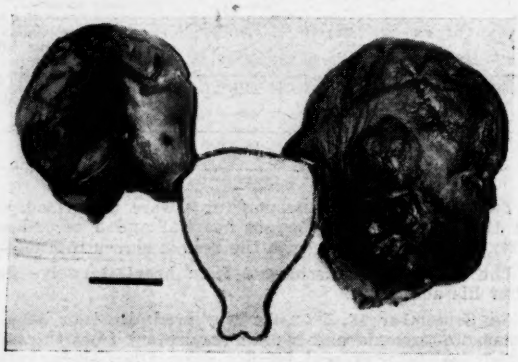

FIGURE I.
Gross appearance of bilateral tumours (the bar measures one inch).

Microscopic examination of one of the tubes revealed some invasion of its wall, but not penetration. Elsewhere, there was a papillary tumour arising from the epithelium of the Fallopian tube (Figure II). The cells were columnar and non-ciliated; they had a centrally placed ovoid nucleus (Figure III), and the cytoplasm stained a uniform pink colour with hæmalum and eosin. The free border of the cells was slightly dome-shaped, and there were numerous mitoses to be seen. The columnar cells were set upon a delicate, branching, vascular connective tissue stroma, sometimes in several layers, but often in only a single layer. The walls of the tubes varied in thickness from place to place. In some areas the wall was thin, while elsewhere it showed a considerable degree of fibrous thickening and was mildly infiltrated with lymphocytes.

Comment.

The main clinical diagnostic features of carcinoma of the Fallopian tube are as follows: (i) It occurs in the fifth decade. (ii) It produces a sero-sanguinous discharge. (iii) It causes colicky pain early. (iv) It can be palpated.

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Legends to Illustrations.

FIGURE II.—Section of Fallopian tube showing papillary carcinoma (×120).

FIGURE III.—Cell detail of carcinoma of Fallopian tube (×400).

Reviews.

The Year Book of Obstetrics and Gynecology (1955-1956 Year Book Series), edited by J. P. Greenhill, B.S., M.D., F.A.C.S.; 1955. Chicago: The Year Book Publishers, Incorporated. 7½" × 5", pp. 544, with 83 illustrations. Price: $6.00.

UNDER the experienced editorship of J. P. Greenhill, this Year Book continues to present a useful account of significant current papers on obstetrics and gynæcology. The section on obstetrics is divided into chapters on pregnancy, labour, the puerperium and the newborn. Subsections in the chapter on pregnancy deal with physiology, abortion, ectopic pregnancy, complications and toxæmia, and those in the chapter on labour relate to general aspects, analgesia and anæsthesia, complications, operative obstetrics and uterine hæmorrhage. The section on gynæcology has chapters on general principles, diagnosis, infertility, operative technique, infections, non-malignant neoplasms, malignant tumours, menstrual disorders and endocrinology. The editorial comments present, in addition to the editor's critical views, references to many other relevant contributions in the current or recent literature. This Year Book will be very useful to the specialist in obstetrics and gynæcology, as well as to general practitioners and others who include these subjects within the range of their practice.

Bone and Bones: Fundamentals of Bone Biology, by Joseph P. Weinmann, M.D., and Harry Sicher, M.D., D.Sc.; Second Edition; 1955. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10" × 7", pp. 508, with 302 illustrations. Price: £7 11s. 3d.

WHEN we reviewed the previous edition of this book, which appeared in 1947, we described it as a storehouse of useful information. This it still is, and the authors have set themselves to bring it up to date. They point out that with the many advances that have taken place in our knowledge of histology and biochemistry, it seems possible for the first time to outline more fully a hypothetical mechanism of bone formation and resorption and to present a basic concept of skeletal growth, especially of the growth of the skull. The first part of the book, which deals with normal structure and growth of bone and bones, has been enlarged by the insertion of a discussion on some of the peculiar features of the otic capsule. To the second part, that on pathology of bone and bones, has been added a discussion of osteoid osteoma and cementoma, of fibrous dysplasia of bone and of some peculiar genetic disturbances of the skeleton. The book is attractively produced and freely illustrated, and may still be described as a work of reference which should be available to anyone who has need of precise information in the field of bone pathology.

Psychological Medicine: A Short Introduction to Psychiatry, by Desmond Curran, M.B., F.R.C.P., D.P.M., and Maurice Partridge, M.A., D.M., D.P.M.; Fourth Edition; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 5½", pp. 416, with 20 illustrations. Price: 21s.

IN "Psychological Medicine" Dr. D. Curran and Dr. M. Partridge present an expanded and extensively revised edition of "Curran and Guttmann". From the opening chapter on "The Ætiology of Mental Illness" the authors have aimed at presenting a sound exposition of their subject, based on clinical experience and refreshingly free from extravagant and untested theories. Symptoms and case taking are covered in a practical manner. The chapter on constitutional anomalies includes both personality deviations and defects, and also a brief but quite adequate account of mental deficiency. In the section on organic mental syndromes, organic neurasthenia characterized by over-sensitiveness, emotionalism and debility is described as a complication of debilitating physical illnesses, also as a component of several organic syndromes. Constitutional neurasthenics are described under "Vulnerable Personalities".

Anxiety states included under "affective disorders" are, in the opinion of the authors, far less common than hysterical elaborations of minor anxiety symptoms and depressive states with anxiety.

Alcoholism is dealt with in twenty pages, and both chemical and psychological remedies receive adequate mention.

In "Peripheral Psychiatry" the authors discuss certain borderlands of psychiatry including so-called psychosomatic states, most of which, in their opinion, are best handled by the general physician. They express a very definite scepticism regarding psychogenesis and psychotherapy in the majority of cases of stammering, sleepwalking, enuresis, tics and disordered sexual function.

There is no special section on the psychiatry of childhood, but in the bibliography the reader will find guidance in further study of this and other aspects of psychiatry.

Special techniques of physical treatment are described with the warning that the medical student cannot be expected to master them any more than he should major surgery. But at least he should know something about the indications for their use. The authors are not impressed with the value of acetylcholine so fervently advocated for anxiety states by Sargant and his colleagues. As regards psychoanalysis, the authors write: "The mystery surrounding it has been enhanced by the use of a special jargon which is difficult to understand."

The student will find that this book provides a sound basis on which to build further experience.

The Thyroid: A Fundamental and Clinical Text, edited by Sidney C. Werner, M.D., Sc.D. (Med.), with sixty contributors; 1955. New York: Paul B. Hoeber, Incorporated. 9½" × 6", pp. 814, with 130 illustrations. Price: $20.00.

IT is seldom that one comes across such a fascinating and unusual book as "The Thyroid", edited by Sidney C. Werner, which is described as a fundamental and clinical text. It is contributed to by sixty writers authoritative on the special items, only one of them being British. It is unique not only in its presentation of subject matter, but in its arrangement, as each contributor reviews and summarizes recent literature from the list of references given at the end of each chapter, so that those interested in any particular aspect of this complex subject are saved the necessity of hunting through libraries. In all, 1723 references are discussed, some very briefly, and the editor sums up the subject matter presented in each chapter. The result is that the errors and banalities of so many text-books are conspicuous by their absence, and the reader is presented with a factual compendium and critical appraisal of modern thought and knowledge. This is particularly evident in the introductory chapters on the physiology and biochemistry of the normal gland, in which recent work on radioactive iodine isotopes has been summarized in a most interesting fashion. This has led to better methods of evaluating compounds for their possible antithyroid activity, as over a thousand such drugs have been investigated, and it is unlikely that as yet the most potent have come into current use. The use of electron microscopy is also helping to a better understanding of cellular cytology and must play an important part in the future elucidation of certain problems. It is pointed out that as yet no perfect laboratory procedure has been devised which will define the level of secretory activity of the thyroid, and to date the uptake or release of radioactive iodine promises its greatest value in differentiating between euthyroid and hyperthyroid activity, but it is not absolute as yet. There is an excellent summary of pathological lesions with first-class illustrations and photomicrographs. The various problems as to advisability of operation are well discussed, as are the indications for and limitations of radioactive iodine in carcinoma of the thyroid.

It is seldom one meets with such composite accounts of the various changes in body tissues generally, including skin and

connective tissues and the ocular manifestations. The chapters on myxœdema and hypothyroidism are most helpful. This is not a book for medical students, as one must have a basic knowledge of the thyroid in general to appreciate to the full the commentaries presented, but to those clinics or individuals dealing with thyroid problems the book will be invaluable. The publishers are to be complimented on their care in giving us a first-class production, well printed and free from errors in both text and illustrations.

The Management of Obstetric Difficulties, by Paul Titus; Fifth Edition; revised by J. Robert Willson, M.D., M.S.; 1955. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 9" × 6½", pp. 738, with 348 illustrations. Price: £6 17s. 6d.

THIS book, revised by Wilson, has almost been rewritten, and should now be renamed "A Textbook of Practical Obstetrics", for no longer is the text confined to obstetric difficulties.

The most recent advances in methods and treatment have been discussed, including the use of the newer antibiotics and the latest classifications of heart disease and anæmia of pregnancy.

The routine use of intravenous therapy for Cæsarean section, the tendency always to repeat a Cæsarean section rather than risk a trial of labour, and the routine packing of a uterus during Cæsarean section when the patient is not in labour would not find universal favour in Australia.

One would feel that the methods of anæsthesia need to be revised when 20% of Cæsarean section deaths are reported as being due to the anæsthetic agent, of which half were given by the spinal route.

It is suggested that artificial rupture of the membranes may reduce the severity of fibrinogenæmia. Bartholomew's views on the cause of bleeding in *placenta prævia* are stated to show that the bleeding comes from the torn marginal sinus rather than directly from the placental site. Australian obstetricians would agree with Clifford's concept of the placental dysfunction syndrome being synonymous with post-maturity rather than the author's experience that it is not a hazardous state for the fœtus to be post-mature.

The illustrations and references are good. However, the book is unlikely to replace the text-books already acceptable in Australia.

Manual of Hand Injuries, by H. Minor Nichols, M.D., with a foreword by Michael L. Mason, M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 352, with 180 illustrations. Price: $9.50.

THIS book is written primarily for students, residents and the surgeon who occasionally has to operate on the injured hand.

The principles of treatment advocated are sound and would meet with almost general approval.

The introductory chapters dealing with the anatomy of the forearm and hand and general technique are standard, but are very easily read. The succeeding chapters discuss amputations, burns, skin grafting, avulsions of skin, tendon and muscle injuries, fractures and dislocations and infections.

The section devoted to secondary tendon repair and tendon grafting is the fruit of much experience. Of interest is tenodesis of the *flexor profundus* to the middle phalanx as a way out of the problem of the intact *sublimis* and a *profundus* incapable of repair. This is probably more certain than the usual arthrodesis of the distal interphalangeal joint, as in quite a proportion of attempted arthrodesis operations failure to fuse occurs.

The last chapter is directed to the simpler elective and reconstructive procedures. The author advocates the removal of any aberrant tendon should it be found during the course of an operation for de Quervain's disease. This is not really necessary so long as the stenosed sheaths are freely incised.

The only other criticisms that could be made are as follows: (*a*) The use of the fluoroscope in the treatment of Colles's fracture, instead of waiting for formal radiographs to give information about the accuracy of the reduction. (*b*) The advocacy of split skin grafts to traumatic amputation stumps. In a book such as this it would probably be wiser to advocate resection of sufficient phalanx to permit primary closure, save for the thumb or multiple amputations. The only significant omission is a full discussion of the management of the unhealed amputation stump.

Premature Infants: A Manual for Physicians, by Ethel C. Dunham, M.D.; Second Edition; 1955. New York: Paul B. Hoeber, Incorporated. 9½" × 6", pp. 474, with 41 illustrations. Price: $8.00.

THIS is the second edition of this work, the first having been published in 1948. It is divided into three parts, namely general considerations, clinical considerations and public health considerations. There is also an appendix concerned mainly with statistics and recommendations for uniform recording and compilation of statistics. The bibliography is very full and helpful and in many cases the original text is quoted at some length. This book is a reference book rather than a manual on the practical day-to-day care of the premature infant.

The general considerations cover the World Health Organization definitions and criteria of prematurity. It is obvious that premature infants must be classified in these accepted four groups: (i) Up to 1000 grammes (2 lb. 3 ozs.); (ii) 1001 to 1500 grammes (2 lb. 4 ozs. to 3 lb. 4 ozs.); (iii) 1501 to 2000 grammes (3 lb. 5 ozs. to 4 lb. 6 ozs.); (iv) 2001 to 2500 grammes (4 lb. 7 ozs. to 5 lb. 8 ozs.). In this section also incidence, causes and prevention of prematurity are considered. In the prevention of toxæmia the ante-natal programme of the Women's Hospital, Crown Street, Sydney, is given. Development and prognosis are also dealt with.

In the clinical considerations the care of the premature infant is discussed in general rather than in detail. The feedings, with preference for cow's milk rather than breast milk, are more complicated than those in general use in Australia. Also, here with our large pool of midwifery nurses capable of feeding infants by gavage, the indwelling naso-gastric tube is not a necessity.

Malformation and abnormalities are dealt with at some length, not only as they affect the premature infants. In discussing retrolental fibroplasia, the author takes a cautious stand on the relationship of retrolental fibroplasia to oxygen administration. We are disappointed to find no reference to the troublesome jaundice of prematurity.

In the past, different methods of recording and keeping statistics have made comparisons of little value. Even in this book, statistics based on information from death certificates, a very doubtful source, are given.

The apposite portions of the publication by the American Academy of Pediatrics, "Standard Recommendations for Hospital Care of Newborn Infants—Fulltime and Premature", are given. It is noted that the New York Health Department pays the expenses of the premature infants of poor parents, over and above the amount the parents can contribute. The average total cost is $320, the average stay in hospital is 27 days, and the parents contribute 3% of the cost.

Not all pædiatricians will agree with the opinions expressed in this book, but all will find useful information and a comprehensive précis of the literature on prematurity.

Ear, Nose and Throat Nursing, by F. Boyes Korkis, M.B., Ch.B. (N.Z.), D.L.O. (Eng.), F.R.C.S. (Ed.), F.R.C.S. (Eng.), F.I.C.S.; 1955. London: J. and A. Churchill, Limited. 8" × 5¼", pp. 176, with 85 illustrations. Price: 12s. 6d. sterling.

THIS book is intended for the student nurse. The first eighty-three pages are theoretical; the anatomy, physiology, pathology and treatment of all common conditions are well described. This will ensure a sound background. Some parts, however, go beyond what most nurses need to know; for instance, the detailed anatomy of the facial nerve.

The final seventy pages provide a chapter each on the out-patients' department, pre-operative and post-operative care, and ear, nose and throat operations. They are truly practical. Noteworthy features are the lists of equipment needed for the usual procedures and exact details of methods which are essentially similar to their counterparts in Australia.

Diagrams illustrate the theoretical chapters, while photographs are mainly used for the practical parts. The former are admirably clear, but the latter sometimes suffer from lack of definition because of inadequate backgrounds.

Otolaryngologists may rest assured that their patients will be well nursed if this book is followed. As a text-book in the

nurses' training school and for ready reference in an ear, nose and throat ward or out-patients' department it is highly recommended. It is more expensive than similar books of recent years, but in subject matter it compares favourably with the best of them.

The First Interview with a Psychiatrist and the Unconscious Psychology of all Interviews, by Charles Berg, M.D. (Lond.), D.P.M.; 1955. London: George Allen and Unwin, Limited. 8½" × 5½", pp. 240. Price: 18s.

IN this book Dr. Charles Berg describes his method of looking beneath the surface of things, of revealing clues to the hidden forces responsible for the patient's symptoms. From his own clinical experience he goes so far as to say that, as with the love-motif in fiction, so in everyday life, sexuality plays some part in every or almost every human relationship, and not least in the doctor-patient situation. Dr. Berg is a Freudian, and accordingly gives a wide interpretation of sexuality, but clearly there can be no deep understanding and confidence in psychotherapeutic relationships without an instinctive basis. Even aggressive trends, the apparent antithesis of sexuality, turn out in the author's experience to be reactions of frustration, the sense of rejection by those who should have loved the organism. "The art of therapy is", writes Dr. Berg, "to supply on a mental plane what mother's love and subsequent human contacts should have supplied at the very beginning of life." The latter part of the book is devoted to descriptions of types, immature men, sexually inhibited men, the socialite woman, the frigid woman and others.

There is a useful glossary of psychiatric and, more particularly, of psychoanalytical terms.

The author writes in an easy style which recapitulates the apparent informality of the interview. But he has not omitted to guide his readers in their approach to more withdrawn psychotic patients. In the chapter on the psychiatric interview we are reminded of details of information, derived from the patient and his relatives, on which the diagnosis must be based. But the patient's life story can seldom be elicited in a systematic manner, and only by experience will the psychiatrist learn how to put leading questions without stirring up the patient's resistance.

Every psychiatrist, though he may not subscribe to theories of Œdipus-complexes and of death-instincts, can glean much from this book about the all-important technique of the first interview.

Psychology in Nursing, by Wendell W. Cruze, Ph.D.; 1955. New York: The Blakiston Division, McGraw-Hill Book Company, Incorporated. 9" × 6", pp. 504, with 79 illustrations. Price: $5.50.

ALTHOUGH written specifically for student nurses, this is essentially a straightforward text-book of psychology at about first year university level. There is an introduction on "How to Study" which is helpful to any student, and there is passing reference in the early part of the book to the value of a knowledge of psychology to the student nurse. Two chapters at the end are devoted to the psychological aspects of nursing children, adolescents, the chronically ill and the aged.

Each chapter commences with a summary and ends with a series of questions and exercises and a list of references which should be a real help to a student. There is a very clear section on genetics, well written and well illustrated; in fact, throughout the book the style is succinct and easy to read and the illustrations are clear and to the point. A description of the normal development of the individual is followed by a very good outline of the structure and the function of the nervous system, with sufficient detail for the student nurse but not overloaded with the more difficult features of neurology.

The motivation of human behaviour, the emotions and their effect on health, an analysis of personality and the development of personality are dealt with very adequately in a comparatively short section.

Professor Cruze must be a very good teacher. The meaning of conflict, frustration and adjustment, rationalization, withdrawal and negativism and similar terms rapidly becomes clear to the reader when presented in Cruze's lucid straightforward style.

The psychoneuroses and psychoses are briefly described, and the point is made that it is dangerous for the amateur psychologist or nurse to attempt to diagnose or treat these conditions.

Other subjects included in the text-book are intelligence and aptitudes, observation and thinking.

The writer does not "talk down" to his student nurses, and there is no doubt that a knowledge of correct scientific psychology would be helpful to nurses, as it is to doctors, teachers and all whose work involves dealing with people. However, to use the book the nurse would require a good educational background, preferably with some science training at school.

Subphrenic Abscess, by H. R. S. Harley, M.S., F.R.C.S.; 1955. Oxford: Blackwell Scientific Publications. 8½" × 5½", pp. 223, with 35 illustrations. Price: 35s.

THIS monograph on subphrenic abscess by H. R. S. Harley appears to be genuinely written for the purpose of lowering the mortality rate of this condition by better understanding and management. The undoubted superiority of extraserous methods of drainage is emphasized in this close study of the records of 188 cases. Peptic ulceration, acute appendicitis and abdominal operations, mainly gastrectomy and biliary surgery, constituted the commonest causes. The author believes that infection to the subphrenic area is nearly always the result of direct intraperitoneal spread.

Recognition of frequent other complications is important and occurred in 64% of the cases in this study; they were mainly serous pleural effusions. Interesting chapters on the clinical features of intrathoracic suppuration and abdominal complications following subphrenic abscess are included. Radiological changes and a plea for screening are well described and emphasized by many illustrations. The diagnosis is established clinically and radiologically and aspiration is condemned because of its unreliability and risk of spreading infection. Exploratory operation is much safer. Chemotherapy will not cure a formed abscess but accelerates resolution after drainage and may allow a "subphrenitis" to resolve without abscess formation.

The technique of drainage is well described. All the spaces, except the lesser sac, can be reached by the posterior or anterior extraserous approaches.

The mortality figure of 40% in the teaching hospitals in London is alarming and in itself makes the reading of this book essential for those called upon to treat this condition.

Diagnosis of Congenital Heart Disease: A Clinical and Technical Study by the Cardiologic Team of the Pediatric Clinic Karolinska Sjukhuset, Stockholm, by Sven R. Kjellberg, Edgar Mannheimer, Ulf Rudhe and Bengt Jonsson; 1955. Chicago: The Year Book Publishers, Incorporated. 10" × 7", pp. 674, with 581 illustrations. Price: $22.00.

A CONSIDERABLE array of monographs, atlases and reference texts dealing with congenital heart disease have appeared in both Europe and the United States during the past ten years. Some have been written by a single author, some by a clinician and pathologist in collaboration, and the best of them by a team of workers which usually includes a radiologist. The most thorough and best illustrated presentation we have yet seen is entitled "Diagnosis of Congenital Heart Disease: A Clinical and Technical Study by the Cardiological Team of the Pediatric Clinic, Caroline Hospital, Stockholm", and records their clinical and röntgenological studies of almost four hundred children over three years. A main object of this study was to make an evaluation of correlative phonocardiography and electrokymography together with the conclusions suggested by clinical evidence and the results of cardiac catheterization. There is abundant evidence that dependence upon clinical signs, plain teleröntgenograms, and cardiac catheterization is by no means always adequate for a final anatomical diagnosis. Each cardiac deformity is described under these five headings, and it is obvious that they have proved the diagnosis in almost every case. The most convincing diagnostic proof is provided by excellent reproductions of high speed angiocardiograms, a technique which seems to have originated in, and remained confined to, Sweden. There is an abundance of such illustrations, in some of which dyed blood can be seen emerging as a jet from one or other type of stenosis. The phonocardiography, while adequate, is less detailed and more coarsely recorded than is possible by the string galvanometer favoured in Great Britain. Reproductions of teleröntgenograms are generous in number, and of high quality. The main abnormalities they purport to show are indicated by small arrows. This is helpful providing

the arrows do not impinge upon the particular abnormality they are intended to indicate. Good illustrations of the actual abnormalities in autopsied hearts are supplied, but congenital heart disease must be studied in all planes in the actual specimen to be convincing and easy to appreciate. While there is little new information in this book, it provides a splendid summary of current views and methods of diagnosis; it is well produced on good art paper, in excellent English, and with a full bibliography. It can confidently be described as the best illustrated monograph on congenital heart disease we have yet seen, and while few clinics can hope to obtain angiocardiograms of this quality without the expensive Swedish electronic equipment, they should each possess a copy for reference and study. The angiocardiograms alone make such a purchase worth while.

Mitchell's Anatomy and Physiology, and Causes of Disease, by H. C. Trowell, O.B.E., M.D., F.R.C.P.; 1955. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 250, with 87 illustrations. Price: 7s. 6d.

THIS book is written by a lecturer in medicine in the Uganda Medical School for "those African nurses and medical assistants who feel the need of a book to introduce them to the study of anatomy and physiology". It probably fulfils their purpose quite well—a particularly high standard is not called for. Some inaccuracies spring to the eye. For example, the epiglottis is not a "lid" to the glottis. It takes no active part in deglutition as is implied on pages 100 and 120. The description of a leucocyte which stuns a bacterium before "eating them" is dramatic, but not strictly accurate. On page 162 we read that part of the uterine wall develops into the placenta; the correct position is stated on page 169, that the embryo develops the placenta. In spite of these and other inaccuracies, we may say that an African nurse or medical assistant who knew this book would be in a position to extend his knowledge without much difficulty. We doubt whether the book will find a sphere of usefulness in this country.

Bedwetting, by Portia Holman, M.A., M.D. (Cantab.), M.R.C.P., D.P.M. (Lond.); 1954. London: Delisle. 7½" × 5", pp. 40. Price: 8s. 3d.

THIS small book is a precise account of the problem of nocturnal enuresis. There are contained no important new ideas, and the language is simple so that by the aid of the provided glossary of medical terms the book may be read with understanding by non-medical persons. There may be some tendency to overplay the conscious resistance of the child to regular urinary continence and also to attach undue importance to psycho-sexual behaviour in later childhood. Otherwise the subject is well and neatly presented and the various organic and functional causes of enuresis are discussed, together with the need for calm reassurance in non-organic cases and the use of conventional drug therapy. The appendices are the most informative part of the book and contain records of such findings as intelligence quotients, organic disabilities and bladder capacity found on the examination of enuretic children. This book may with profit be recommended to nurses and midwives and to the anxious and intelligent parent.

A Short Textbook of Midwifery, by G. F. Gibberd, M.B., M.S. (Lond.), F.R.C.S. (Eng.), F.R.C.O.G.; Sixth Edition; 1955. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 604, with 199 illustrations. Price: 30s.

THIS, the sixth edition of "A Short Textbook on Midwifery", by Gibberd, follows the general form of previous editions with revised sections on toxæmias, uterine inertia and ante-partum hæmorrhage.

The early chapters of the book contain sound information on the anatomy and physiology of pregnancy and labour. The precautionary measures to be taken against sepsis are discussed in great detail and are of interest in view of the excellent, although fortunately largely outdated, section on puerperal infections appearing later.

The prevention and early detection of pre-eclamptic toxæmia are considered at length and a section on treatment follows. Here it is felt that a conservative attitude has resulted in a critical rather than a constructive approach to these problems. The indications for termination of pregnancy in this condition, however, are enunciated quite simply.

Early and thorough investigation of delay in the first stage of labour is urged so that serious associated abnormalities may be detected or excluded. The place of sedation, oxytocic drugs and Cæsarean section is discussed at some length.

The summary of the management of *placenta prævia* conforms to modern teachings, whereas that of accidental hæmorrhage is essentially conservative.

This conservatism is the keynote of the volume and no doubt reflects the personal views of the author. Much that is practised elsewhere, for example, pudendal block anæsthesia, is omitted. Other subjects, such as the place of anticoagulant therapy, are left in doubt as to recommendations and detail. In spite of these criticisms the book is sound in its essentials and a useful short reference.

Management of Pain in Cancer, by Reuben C. Balagot, M.D., J. W. J. Carpender, M.D., E. G. Gross, Ph.D., M.D., Max S. Sadove, M.D., Henry Schwarz II, M.D., Oscar Sugar, Ph.D., M.D., Samuel G. Taylor III, M.D., and V. Richard Zarling, M.D. Edited by M. J. Schiffrin; 1956. Chicago: The Year Book Publishers, Incorporated. 7" × 4½", pp. 245, with illustrations. Price: $4.50.

THIS small book provides in a compact form a review of the methods available for palliation of the sufferings of the patient with cancer. It should prove very useful, on account of the additions which have been made in recent years to our armamentarium. The various sections have been written by the appropriate specialists and they describe clearly and concisely the use of drugs, surgical and neurosurgical methods, and radiotherapy, in the treatment of pain. Especially welcome is the account of the new hormonal methods in the treatment of mammary and prostatic carcinoma and the manner in which they fit into the general scheme of treatment. The practical tone of the book is illustrated by the following advice. "Oophorectomy is indicated (in breast cancer) in the menstruant at the first sign of visceral or osseous metastatic disease. Following failure of this procedure androgens could be tried." In allotting this priority the authors remark that "put to the vote, most women would rather have hot flushes and dizzy spells than whiskers and a male voice".

The various cytotoxic drugs at present being tried are briefly dealt with, and the book concludes with a useful chapter on the psychological aspect.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A History of the Therapy of Tuberculosis and the Case of Frédéric Chopin", by Esmond R. Long, M.D.; Logan Clendening Lectures on the History and Philosophy of Medicine; Sixth Series; 1956. Lawrence: University of Kansas Press. 8½" × 5½", pp. 75. Price: $2.00.

Chopin is introduced as an example of the helplessness of other days.

"The Medical Clinics of North America"; 1956. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. Memorial Center Number. 9" × 6", pp. 983, with illustrations. Price: £8 2s. 6d. per year in cloth binding and £7 15s. per year in paper binding.

Consists of a symposium on the medical aspects of cancer.

"The Year Book of Endocrinology (1955-1956 Year Book Series)", edited by Gilbert S. Gordon, M.D., Ph.D.; 1956. Chicago: The Year Book Publishers, Incorporated. 7½" × 5", pp. 367, with many illustrations. Price: $6.00.

One of the "Practical Medicine Series" of Year Books.

"The Year Book of Dermatology and Syphilology (1955-1956 Year Book Series)", edited by Rudolf L. Baer, M.D., and Victor H. Witten, M.D.; 1956. Chicago: The Year Book Publishers, Incorporated. 7½" × 5", pp. 480, with many illustrations. Price: $6.50.

One of the "Practical Medicine Series" of Year Books.

"Neurology and Psychiatry in Childhood", Proceedings of the Association, December 10 and 11, 1954, New York, N.Y. Research Publications, Association for Research in Nervous and Mental Disease, Volume XXXIV. Editors: Rustin McIntosh, M.D., and Clarence C. Hare, M.D.; 1954. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 517, with many illustrations. Price: £6 1s.

Consists of six parts, each of which is divided into four or more sections.

The Medical Journal of Australia

SATURDAY, AUGUST 25, 1956.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words of phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

TELLING THE TRUTH THROUGH DRUGS.

THE well-known saying "*in vino veritas*", which originated with Pliny, reminds us that alcohol loosens the tongue and enlarges the scope of conversation. We know that when a man is "in his cups" he is likely to say things and reveal secrets to his acquaintances, his friends and even those closely related to him, which in the ordinary course of events he would rather keep hidden from them. Alcohol is not the only drug which has such an effect, and this fact has been used in the detection of crime. At first sight this may appear to be an admirable and entirely useful expedient, but serious thought needs to be given to its effectiveness and to the ethics of its use.

One of the latest advocates of what he calls "narco-interrogation" is C. B. Hanscom,[1] Director of the Department of Protection and Investigation of the University of Minnesota. He describes his introduction to the method. Previously he had used only the polygraph, but in September, 1948, he was faced with the following case. A young man with a long police record was charged with the brutal murder of a boy and a girl whom he had surprised in unusual circumstances on a golf course. The man's conviction finally depended on the location of the murder rifle. Polygraph examination disclosed his guilt and he reacted to any possible location of the rifle which involved a body of water—the correct river, lake or pond could not be selected. "In desperation" they turned to the only other method of investigation which had not been used—interrogation while the suspect was influenced by anæsthetic drugs. After three or four hours of questioning a complete confession of the crime was obtained with a description of the location of the gun; a post-hypnotic suggestion was made that the suspect would lead his questioners to the spot from which he threw the weapon into the pond. The suspect did help to locate the rifle and "the case was prosecuted successfully". Since this happening more than thirty different tests have been made under narcosis, and Hanscom writes: "They have been concluded to our satisfaction and validated by the facts subsequently disclosed. We have no evidence of a failure in the series." Hanscom writes that, though the final theory of narcosis is unexplained, clinical evidence of the anæsthetic stages indicates that the various segments of the brain may be selectively depressed in a known order corresponding to their evolutional age, the more recently acquired cortex being depressed first. It is believed that the cortex performs the discriminatory and integrative actions of the personality, while the diencephalon expresses the primitive emotional drives. In narco-analysis for criminal interrogation the depression of only the cortex and diencephalon is needed and this can be achieved in the first and second stages of general anæsthesia. Hanscom continues: ". . . by variation in the level of depression and selective stimulation, combined with proper interrogation techniques, we can modify the personality functions and lead the suspect into known confession mechanisms." He enlarges on this statement in a way which cannot be reproduced here. Hanscom obtains the consent of the subjects to the procedure; he states that they consent because refusal might indicate complicity, or because they believe they can avoid detection and strengthen their case in court—they "have little knowledge of the successful record of the technique". In another place he states that the suspect definitely understands his constitutional liberties, which allow him to refuse the examination or accept the possibility that its results might be used in subsequent investigations or prosecution. One of his general conclusions is that drug-induced statements must be corroborated by facts before they can serve defence or prosecution in court. Hanscom describes the technique which he uses—it cannot be described in this place. He states that acts committed during profound alcoholic stupor and associated with severe head wounds are probably not accessible through this means. He also makes the important statement that no reports have been discovered to the effect that narcoanalysis has been used to force a suspect to confess a crime he did not commit. This is of the greatest importance, for the possibility of such an event is one of the strongest objections to the use of the method. Although there is no actual evidence to support the idea, it is reasonable to surmise that the greater number of reported confessions of guilt from persons of high repute that come from behind "the iron curtain" are obtained through the medium of drugs aided by suggestion, perhaps of the most violent kind. Be that as it may, attention must be drawn to a paper by John M. Macdonald, Associate Professor of Psychiatry in the University of Colorado School of Medicine, published in the journal that contains Hanscom's article. Macdonald declares that examination with the aid of drugs does not always lead to the truth, and that persons, while under the influence of drugs, may show increased susceptibility to suggestion and false confessions of guilt may be made. He reports cases from the literature to prove this. He

[1] *J. Forensic Med.*, January-March, 1956.

states that Ripley and Wolf noted that some patients while under the influence of "Sodium Amytal" deliberately misrepresented facts. He also mentions an experiment reported by Redlich *et alii* in which nine volunteers were asked to maintain artificial lies in a "Sodium Amytal" interview—three of the nine were able to continue the deception under the influence of the drug. Macdonald summarizes the situation by stating that criminal suspects, while under the influence of barbiturate drugs, may deliberately withhold information, persist in giving untruthful answers or falsely confess to crimes they have not committed. Narcoanalysis, he thinks, is likely to be effective only in cases of hysterical amnesia or in persons who would be likely to confess anyway without the use of drugs. Therefore, he believes that its usefulness to law-enforcement agencies is confined to persons suffering from hysterical amnesia; it should not be used as a substitute for skilled interrogation by competent police officers. The one other sphere in which he will allow that it may be useful is in the psychiatric examination of suspected criminals who have entered a plea of not guilty because of insanity. How shall we judge the effectiveness of this method when we have the caution of Macdonald on the one hand and the enthusiasm of Hanscom on the other? The conditions of the testing described by the latter appear to be fair to the suspect, but a very great deal will depend on the type of interrogation—the questions asked and the way in which they are asked. At the same time it is beyond doubt from the literature that suggestion is most likely to be operative. Of the power of suggestion in oral examination no experienced clinician can have any doubt, and the zealous police officer has probably yet to be born who could avoid it with the suspected individual more or less at his mercy. On the available evidence the verdict must be that the general effectiveness of this method of examination is not proven.

What of its ethics? As the business has to do with the use of drugs, the question is of the greatest importance to the medical profession. Those concerned will be chiefly prison or police medical officers or anæsthetists called in specially for the work. At first sight one might argue that if, as Hanscom does, one gives the subject the right to refuse to submit to the examination, ethical considerations are beyond question. But the subject does not know how effective the ordeal may be; in fact Hanscom, as we have seen, makes a great point of this—the subject has something put over him. Consent therefore will not wipe the ethical slate clean. Macdonald discusses the question whether a medical practitioner should, on the request of the police, administer drugs for the purpose of obtaining a confession. He quotes the view of Lorenz that the practice places the physician in a rather uncomfortable position, because through his medical knowledge and efforts a person is rendered more or less helpless. He refers to the legal position in England as defined by Lord Riddel to the effect that a doctor shares with other citizens the duty to assist in the detection and arrest of a person who has committed a serious crime. For ordinary purposes all practitioners will agree, but the position is surely different if in the ordinary course of his medical attendance on a patient, a doctor discovers that the latter has been engaged in crime. The Declaration of Geneva states *inter alia*: "I will respect the secrets that are confided in me." It is true that a medical man may be compelled by a judge in open court to reveal facts which in his (the doctor's) opinion are covered by the professional seal. The doctor's position is then clear—he speaks under protest and no blame can attach to him. It is only right to point out that the view of Lord Riddel, as stated here, is not always accepted. Saundby, in his standard text-book on medical ethics, refers to an opinion given to the Royal College of Physicians in 1896 by Sir Edward Clarke and Mr. Horace Avory to the effect that a medical man should not reveal facts which had come to his knowledge in the course of his professional duties, even in so extreme a case as where there were grounds to suspect that a criminal offence had been committed. Macdonald writes that a physician who agrees to administer a drug to a criminal suspect on the request of the police should first obtain the suspect's written consent. "The prisoner should be given an opportunity to consult an attorney and should be informed that he cannot be compelled to submit to examination while under the influence of drugs. Furthermore, the physician should confine himself to administering the drug and should take no part in the criminal interrogation. He should intervene only to check on the suspect's physical condition or to protect the suspect from possible coercion by the police." The conclusion is that most medical practitioners will abide by the opinion of Clarke and Avory and will avoid complicity in this kind of investigation to the best of their ability. They will consent to the statement of Guttmacher and Weihofen, quoted by Macdonald from their text-book, "Psychiatry and the Law":

> We submit that the possible injustice that might be done by the suppression of evidence in individual cases is outweighed by the importance of assuring patients that the confidentiality of their relations with the psychiatrist is absolute, and not subject to violation even on a court summons.

One point remains—the ethics of the procedure as a whole. We have seen that it is not fully effective. We have argued that the medical man will best serve his own conscience and medicine in general if he has nothing to do with this kind of examination. In such circumstances we cannot regard this type of examination as strictly ethical. Somehow it does not sound like what we have learned to expect of British justice.

Current Comment.

MUSCLE DYSTROPHY IN CHILDHOOD.

THE muscle dystrophies of childhood belong to the very complex group of heredo-familial diseases, the ætiology of which remains obscure. Between the different classified myopathies there is much similarity, but the syndromes appear to be inherited as intact traits so that the classification into different clinical diseases is justified. According to C. O. Carter,[1] the conditions known as *dystrophia myotonica*, *myotonica congenita*, and progressive muscular atrophy are transmitted as autosomal dominant genes, while the conditions known as progressive muscular dystrophy and hypertrophic muscular dystrophy are transmitted as autosomal recessive genes.

Of all the myopathies, the pseudo-hypertrophic, the scapulo-humeral of Erb, and the facio-scapulo-humeral

[1] "Pædiatrics", Vol. I. Edited by Gaisford and Lightwood; Butterworth, London, 1953.

types are the commonest and the best known. F. E. Stephens[1] has suggested that most cases of progressive muscular dystrophy can be classified into two general groups, the facio-scapulo-humeral type and the childhood type. However, this classification, though simple, does not include the various muscular dystrophies which arise later in life associated with metabolic disorders or even, as H. J. Anderson[2] *et alii* have shown, with conditions such as carcinoma of the bronchus. Attempts to explain the ætiology of the familial muscular dystrophies on the grounds of biochemical changes have also not been satisfactory. Differences in local biochemistry of the affected muscle have been due to degeneration, as pointed out by W. G. Wyllie[3] in the case of creatine and creatinine.

The progression in the various diseases varies widely from subject to subject, and the ætiology of the pseudohypertrophy has not been elucidated. F. H. Tyler[4] concluded that pseudohypertrophy was dependent on an intact lower motor neuron unit. Tyler and M. M. Wintrobe[5] had already separated the muscle dystrophies into the two groups later discussed by Stephens,[1] and they considered that the defect was quite possibly one of disturbed biochemical metabolism in the involved muscle, though in a later study Tyler, together with G. T. Perkoff,[6] reported that no disturbance in biochemical metabolism could be detected.

G. H. Fetterman, M. J. Wratney, J. S. Donaldson and T. S. Danowski[7] have reported the clinical and biopsy findings in a series of 31 children with muscular dystrophy. The incidence of dystrophy in relatives other than siblings was in their series lower than that reported by other investigators. The age of onset was between two and ten years. No abnormalities of the cerebro-spinal fluid were detected. With one exception, all of the muscle groups tested in these children were of impaired efficiency; the changes were symmetrical and were more pronounced in the lower extremities. Biopsy of affected muscle tissue frequently revealed the presence of swollen, degenerated, or atrophic individual muscle fibres lying intermingled with normal muscle fibres. This finding is in contrast with the orderly patterns of neuropathic myopathies in which changes occur in motor units. All types of muscle degeneration and fibrosis of interstitial connective tissue were found. No primary inflammation was observed and no nerve changes were demonstrable. There was no evidence that muscle biopsy produced any harmful effect on the subsequent strength of the muscle as a whole. An attempt to correlate histological and clinical findings revealed that though tissue changes were far advanced in patients with the most pronounced muscle disability, the histological changes might precede the loss of muscular strength.

B. Girdany and T. S. Danowski[8] have correlated the radiological findings in the long bones, spine and soft tissues with the severity of the disease in patients with muscle dystrophy. Lordosis of the spine occurred as a compensatory mechanism both early and late in the disease, while scoliosis occurred more frequently and more severely with the involvement of muscles in the dystrophy. An apparent increase in the altitude of the lumbar vertebral bodies, as seen by lateral radiographs, is a non-specific change seen in chronically bed-ridden children, and is probably due to rotation of the vertebral bodies with apparent decrease in width. Narrowing or constriction of the shafts of the long bones, with remoulding of the metaphyses, was a particularly common finding in the humerus, femur, tibia and fibula. Bilateral *coxa valga* deformities were present in most patients. Radiological evidence of fatty infiltration of the calf muscles was found in advanced cases of the disease, and there was abnormal fatty infiltration in the soft tissues of the upper extremities and the thighs. There were no bony changes in the skull, and the heart and lungs appeared to be normal in each case. There was a tendency to retardation of skeletal maturation and to variable osteoporosis. None of the many radiological abnormalities reported by other authors as specific to muscle dystrophy was found in this series.

T. S. Danowski, P. M. Wirth, M. H. Leinberger, L. A. Randall and J. H. Peters[1] have studied the biochemical aspects of muscular dystrophy in childhood. No specific abnormalities of the blood were found, and studies of kidney and liver function revealed no consistent abnormality. However, serum phosphorus and calcium values tended to be high in muscular dystrophy, and cholesterol and chloride values tended to be depressed, though these abnormalities may have been related to such factors as malnutrition, delayed growth and immobilization rather than to the muscle dystrophy itself.

T. S. Danowski, R. M. Basiani, F. O. McWilliams, F. M. Mateer and L. Greenman[2] in a study of the endocrine metabolism of dystrophic children found that there were two abnormalities. The first was that serum protein-bound iodine values were higher in some children with muscle dystrophy. The evidence indicated that this was due either to increase in the production of thyroid hormone or to an increased binding capacity of the serum proteins. The second abnormal finding was that of decreased acceleration of glucose metabolism in response to insulin. Danowski *et alii* concluded that this was presumably a reflection of the diminution in muscle mass or its involvement by the dystrophic process. Glucose tolerance tests gave normal results, and no abnormality was found in the measurement of serum corticoids and of urinary gonadotrophin and 17-ketosteroid excretion.

There is no specific treatment for the dystrophic conditions of childhood, and as the disease appears to be self-limiting, the problem is one of conserving maximal activity in residual muscle combined with the prevention of deformity. Whether the ætiology will ever be fully understood, and whether a specific treatment will ever be found, only the passage of time can tell.

[1] *Am. J. Med.*, October, 1953.
[2] *Lancet*, December 19, 1953.
[3] "Diseases of Children." Edited by A. Moncrieff and P. Evans; Edward Arnold, London, 1953.
[4] *Arch. Neurol. & Psychiat.*, March, 1950.
[5] *Ann. Int. Med.*, January, 1950.
[6] *Arch. Int. Med.*, August, 1951.
[7] *J. Dis. Child.*, April, 1956.
[8] *J. Dis. Child.*, April, 1956.

THE USE OF CORTISONE IN CHILDHOOD.

WHILE the use of cortisone in the treatment of adults is attended by numerous well-known complications, the use of cortisone for children may, in addition, result in particular side effects related to interference in the normal progression of body growth. Cortisone has been used in various disorders of childhood including *erythema multiforme*, infantile eczema, the nephrotic syndrome, Still's disease, various eye diseases, and the blood dyscrasias, especially of the Schönlein-Henoch type. The main indications for the use of cortisone in childhood occur in the presence of adrenal insufficiency and in hyperadrenia. While the use of cortisone in Addison's disease is logical, it seems paradoxical that cortisone should be used when the 17-ketosteroids are in excess. However, L. Wilkins *et alii*[3] showed that cortisone given over long periods successfully depresses adrenal over-activity so that the administration results in the slowing of growth and of osseous development and in the reduction of 17-ketosteroid excretion. Thus cortisone is used with success in the treatment of the adreno-genital syndrome and in the adreno-cortical or Cushing's syndrome. Insufficient time has elapsed to assay the final outcome of a child reared on graduated doses of cortisone, and D. Hubble and J. H. Hutchison[4] suggest that in addition to the therapy by cortisone the adrenal tumour should be removed whenever possible. They point out that in the use of cortisone in

[1] *J. Dis. Child.*, April, 1956.
[2] *J. Dis. Child.*, April, 1956.
[3] *J. Clin. Endocrinol.*, December, 1952.
[4] "Pædiatrics", Vol II. Edited by Gaisford and Lightwood; Butterworth, London, 1954.

hyperadrenia the rate of growth and of osseous development must not be slowed too much, and that these developmental changes may be used as a means of estimating cortisone requirements. The effective use of cortisone in adrenal hyperplasia in childhood has since been reported by A. Prader,[1] who suggests that cortisone is not merely *hormone suppressive, but also hormone substitutional.* Provided that careful watch is kept on the patient's metabolism and development, cortisone is safe and effective. Using a new, longer-acting depot type of cortisone, Prader[2] has continued to obtain good results in the treatment of adrenocortical hyperplasia.

The effect of cortisone upon testicular development has been reported by L. Wilkins and J. Cara.[3] In their series were seven young boys each with congenital adrenal hyperplasia. In each case prior to treatment, the testes were of pre-adolescent size, and biopsies revealed the immature state of the tubules and germ cells. No change was noted after cortisone had been given to boys whose osseous maturity was less than that of the normal age of eleven years. However, in boys with osseous maturity beyond the normal eleven year stage, cortisone resulted in the rapid growth of the testes with active spermatogenesis and the appearance of numerous Leydig cells. The same phenomenon of different effect according to osseous maturity was seen when treatment consisted of removal of the adrenal tumour. From this observation, Wilkins and Cara concluded that the testicular maturation depended on the rebound release of pituitary gonadotrophins resulting from the suppression of adrenal androgens and œstrogens by cortisone. This activity of the pituitary begins only at an age corresponding to osseous maturity of eleven to thirteen years.

The use of cortisone in female pseudohermaphroditism has also received favourable reports. R. Vines and L. Dods,[4] in the treatment of six such children, observed that with cortisone the 17-ketosteroid excretion was depressed and virilism was either lost or did not develop. The use of cortisone in the treatment of two infants was not without difficulty, and in one child salt metabolism needed correction; in the other additional treatment with desoxycorticosterone was necessary. Vines and Dods found that the maximal suppression of 17-ketosteroids was unnecessary in the attainment of a satisfactory clinical response.

However, the use of cortisone in the treatment of children is not without dangers, additional to the side effects reported by these various authors. J. Luder[5] found four children with vertebral collapse who were receiving cortisone; one was suffering from chloroma and three suffered from rheumatic arthritis. Collapse occurred from six weeks to a year after the commencement of therapy, and changes in the lower thoracic and lumbar vertebræ were found to be similar to those of Cushing's syndrome. Luder considered that the change followed protein loss from the bony matrix as part of a general negative nitrogen balance. Pain was present in only one of the cases, so that frequent radiological examination of the spine of children receiving cortisone therapy was suggested.

The effects of prolonged cortisone therapy on the growth, metabolism and skeletal maturation of children have been more extensively explored by F. M. Blodgett, L. Burgin, D. Iezzoni, D. Gribetz and N. B. Talbot.[6] Children receiving cortisone for various complaints were patients of the Massachusetts General Hospital. In each case the cortisone was administered by mouth and the diet was not restricted. Measurements were made at intervals over four years, together with bone age estimations and electrocardiographic and biochemical estimations. In the group of children with congenital adrenocortical virilism the average rate of growth was far above the normal before treatment. With cortisone, the growth rates and skeletal maturation rates were reduced and it was found that approximately 35 milligrammes of cortisone per square metre of body surface area per day were required to reduce the growth and skeletal maturation rates to normal values. In children with hypopituitarism, only 4 to 20 milligrammes of cortisone per square metre per day were required to suppress growth to any noticeable extent, while such doses tended to increase growth in adrenocortical virilism and Addison's disease. In endocrine-normal children the amount of cortisone needed to affect growth rate to any noticeable extent was 60 milligrammes per square metre of body surface per day. In three patients of endocrine-normal type, growth was slowed by the cortisone, as generally found, but reduction in the dosage to approximately 30 milligrammes per square metre of body surface area per day was followed by a major increase of growth, indicating that cortisone therapy at this level in the endocrine-normal child is not growth suppressive. However, this compensatory spurt was not found in hypopituitary children, and Blodgett *et alii* wonder whether this indicates the storage by the normal pituitary of potentially active secretions during cortisone administration. Other observations revealed that the ratio of height age to bone age tended to fall sharply in the first few years of the lives of untreated children with congenital adrenocortical virilism, the ratio subsequently rising towards normal levels. This dip in values could be corrected only if treatment was begun with cortisone in early infancy. No significant change was encountered in the ratio of height age to bone age in the endocrine-normal children. With more than 50 milligrammes of cortisone per square metre per day there was a tendency, specially noticed in the endocrine-normal children, to increased appetite and increased weight. The same thing occurred with hypopituitary children on a far smaller dosage. Breast development occurred in several young girls. Two endocrine-normal children developed hyponatræmia and one child with adrenocortical virilism had hypernatræmia; one of the former children also developed abnormal values of serum potassium and blood sugar and depressed electrocardiographic *T* waves. The serum alkaline phosphatase level was lowered in two other children. With these exceptions no abnormalities of serum sodium, potassium, calcium, chloride, carbon dioxide, phosphorus, phosphatase, sugar or protein-bound iodine values were found. With the single exception, the electrocardiograms were normal, and in no case was there any radiologically detectable abnormality of the spine.

This survey is of particular value in laying down a fairly precise level of cortisone required to inhibit growth and skeletal maturation. In the dosages employed, the children with Addison's disease are least affected, while those with hypopituitarism are, as might be expected, the most sensitive. In adrenocortical virilism the dosage of cortisone is that needed to keep growth at a normal rate. Finally, for children receiving cortisone for diseases not of endocrine origin, it would appear to be advisable to reduce dosages at intervals to allow the compensatory acceleration of growth and skeletal maturation to take place and thus to attain the average normal growth and maturity.

[1] *Helvet. pædiat. acta*, November, 1953.
[2] *Acta endocrinol.*, 14, 1953.
[3] *J. Clin. Endocrinol.*, March, 1954.
[4] *Australasian Ann. Med.*, February, 1954.
[5] *Great Ormond Street J.*, June, 1954.
[6] *New England J. Med.*, April 5, 1956.

N-ACETYL-P-AMINOPHENOL IN PÆDIATRIC PRACTICE.

N-ACETYL-P-AMINOPHENOL is a compound related to the salicylates and appears to arise during the metabolism in the body of acetanilid and acetophenetidin; it is probably the major active constituent of such compounds occurring as a metabolite. The pharmacological and clinical properties of n-acetyl-p-aminophenol have been studied for some years, and while various precise conclusions have been reached, no widespread attempt has been made to substitute it in the conditions normally treated by the conventional salicylates. The antipyretic and analgesic efficiency of n-acetyl-p-aminophenol has

been established. The toxicity appears to be relatively low and the LD50 is rather more than twice that of acetylsalicylic acid. Prolonged treatment has been found to affect neither the bone marrow function nor the biochemistry of the blood and urine. Absorption after administration by mouth is rapid; peak plasma levels are said to be reached after one to two hours, and most of the compound is recovered from the urine in a conjugated form. No aniline or aniline derivative is produced. Various investigators have shown that methæmoglobin is not produced; anæmia, the depression of bone marrow function, and undesirable effects on the blood clotting mechanism have not been demonstrated.

D. A. Cornely and J. A. Ritter[1] have made a clinical trial of the effectiveness of n-acetyl-p-aminophenol in the treatment of children, using the compound made up in the form of an elixir. The compound was used in the treatment of 121 infants and children suffering from pain, fever and irritability attributable to a variety of causes. Antibiotics and chemotherapeutic agents were also administered when indicated to 96 of the patients. Of the 20 children who received only the new compound, a good antipyretic effect was noted in 18. A good analgesic effect was obtained in 68 of the whole group of children, and the only possibly attributable side effects were gastro-intestinal upsets in five patients. The compound was also used in the treatment of 20 children with tuberculosis and was administered over a period of five weeks with a rest period of one week after each two weeks of treatment. Each of these patients was examined carefully for signs of interference with the normal physiology of the blood, but no side effects of this nature were found. Cornely and Ritter conclude from their study that n-acetyl-p-aminophenol was considered to be "acceptable" or "liked" by 122 of the 141 children who received it. The authors were also able to suggest a schedule of dosage of the new compound according to the age of the child. This dosage appears to be fairly high in view of the non-rheumatic nature of the illnesses treated. From the lack of controls it is impossible to make any comparison between the new compound on the conventional salicylates or between the new compound and no salicylate. The difficulties of evaluating the subjective effects of drug treatment leave the conclusions to be little more than an impression gained by particular workers in this limited series. The question of whether it is justifiable to administer antipyretic analgesic drugs as a routine in childhood illness is not discussed. However, the compound appears to be safe and well tolerated, and further investigations may well reveal n-acetyl-p-aminophenol to be a useful drug in the treatment of acute and chronic rheumatic states.

SPONTANEOUS SPINAL SUBARACHNOID HÆMORRHAGE.

SPONTANEOUS intracranial subarachnoid hæmorrhage is a comparatively rare disaster, which unfortunately usually affects younger people. The hæmorrhage is usually the sequel to degenerative changes into, or unusual stress thrown upon, the walls of a congenitally abnormal blood vessel. The circle of Willis is the most common site of aneurysms which lead to eventual intracranial hæmorrhage beneath the arachnoid membrane. Interest in these abnormalities has increased since the advent of radiological techniques designed to demonstrate the lesion in the living subject. Neurosurgical intervention may successfully stop the hæmorrhage, or prevent it by obliterating or reducing the particular circulation at risk. The less common, but equally perilous, spontaneous spinal subarachnoid hæmorrhage has attracted less attention despite the findings of such workers as Brion *et alii*,[2] who revealed that a surprisingly high proportion of vascular malformations of the spinal cord was discovered in the routine post-mortem examination of subjects who had exhibited signs of myelitis. Certainly, such abnormalities of the spinal cord as angioma and other highly vascular neoplasms are by no means rare. Likewise, any disease of the blood vessels or any dyscrasia leading to spontaneous bleeding may result on occasions in spontaneous subarachnoid hæmorrhage in the spine. Typically, the patient experiences sudden pain at the site of bleeding, with radiation of the pain to the sciatic region and with signs of meningeal irritation. Difficulty is experienced in diagnosis, particularly if the hæmorrhage is in the cervical region, as the signs and symptoms may closely resemble those of intracranial subarachnoid bleeding. R. A. Henson and P. B. Croft[1] report that in a group of 12 such cases there was a common finding of symptoms of intracranial extension of the hæmorrhage supervening at a variable interval, depending upon the level of bleeding in the spine. Serious disturbance of spinal cord function may result from hæmatomyelia. Otherwise, the spinal cord and nerve roots can be compressed by either blood or blood clot, and organization of the latter may result in continued compression of the spinal cord after recovery from the initial hæmorrhage. In the cases described by Henson and Croft neurological signs of lesions of the cord were variable and in three patients were absent. In two of the patients who died, hæmorrhage was present in both the intracranial and spinal areas, and the primary lesion was one of *polyarteritis nodosa*. In patients who recover, the cause of the bleeding may be obscure, but it would appear that spinal angiomata are the commonest lesions present. The diagnosis of spinal subarachnoid hæmorrhage is made on the history and on the findings in the cerebro-spinal fluid. In cases of small hæmorrhage, the condition may be confused with a lesion of an intervertebral disk. Moreover, confusion may occasionally occur in leaking intracranial vessels accompanied by only low backache and sciatic irritation. Some help is given in the diagnosis of angioma of the spinal cord by the occasional concomitant vascular nævi found in the skin as congenital abnormalities. Obviously, lumbar puncture is not without risk in cases of angioma of the lumbar region, though demonstration of a spinal block and visualization of the lesion by myelography are particularly useful. Aneurysms occur rarely in the spinal canal and are unlikely to be diagnosed in the live patient. The hæmorrhage from neoplasms appears to be relatively slow, but recurrence is most likely in these cases. In this series the five patients who survived made good recoveries. Hanson and Croft suggest that treatment of spinal subarachnoid hæmorrhage is advisedly conservative; they were unable to reach any conclusions on the therapeutic value of lumbar puncture. They consider that no measure is likely to be of avail in massive hæmorrhage, though surgical treatment of spinal compression caused by organized blood clot or by arachnoidal adhesions may occasionally be indicated.

[1] *J.A.M.A.*, April 7, 1956.
[2] *Arch. Neurol. & Psychiat.*, September, 1952.

EXPERIMENTAL MONOCYTIC LEUCHÆMIA.

WHAT appears to be a remarkably specific action on the production and proliferation of monocytes, often with abnormal nuclei, has been announced from Rome by Francesco Pentimalli.[2] Rabbits of 2·0 kilograms average weight were repeatedly injected with a heterologous protein, the most efficacious being lactoglobulin, lactalbumin and egg albumen; whole cow's milk in a fresh state gave excellent results. The increases in leucocyte count were high, from 8000 per cubic millimetre to 30,000 or 40,000, monocytes and monoblasts preponderating. The reticuloendothelial system was chiefly affected and this gave rise to cells of the monocyte type often pathological in grade of maturity, nuclear shape and abnormal mitosis; these multiplied in the circulation until an easily detected leuchæmia was produced. It is indeed remarkable that a heterologous protein should single out the monocyte precursor cells and leave other systems undisturbed. As leuchæmogenesis is regarded as allied to carcinogenesis, the importance of this observation is considerable.

[1] *Quart. J. Med.*, January, 1956.
[2] *Science*, March 16, 1956.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Normal and Immune Hæmagglutinin.

K. B. FRASER (*J. Path. & Bact.*, July, 1955) has considered the formation of antibody. He began by making a study of the relationship between a normal and an immune hæmagglutinin. He chose the system of T. agglutinins against R.D.E. treated human red cells. The standard agglutinating serum was the serum of the donor of the red cells. This naturally occurring antibody had a constant titre of one in 64. The immune sera were prepared in rabbits by the injection of 0·5 millilitre of packed red cells in 2·0 millilitres of fluid given every seven days for six to nine doses. Standard agglutination tests were carried out, and these were read after one hour at 37° C. and after a further two hours at 4° C. The normal T. agglutinin acted best at 37° C.; absorption of this agglutinin at 37° C. reduced the titre at 4° C. to cells from another species. When tested with R.D.E. treated cells there appeared to be differences; one pattern was constant for the treated, the other for normal untreated cells. There was evidence to show that the normal antibody becomes altered in its character by immunization. In two animals, tested after a lapse of time, the modified antibody reverted to its normal character. The author concludes that the normal antibody-producing mechanism may be modified by the introduction of a new antigen, and that the results are better explained by Burnet's postulate of adaptive enzyme formation than by the Haurowitz and Mudd theory of template specific complementary structure.

Virulent and Avirulent Staphylococci.

J. M. SMITH AND R. J. DUBOS (*J. Exper. Med.*, January, 1956) studied the behaviour of virulent and avirulent staphylococci in the tissues of normal mice. Two strains of *Staphylococcus pyogenes* coagulase positive, phage type 7·47C and 44A.44E, from patients with osteomyelitis, phage type 52A, two strains of coagulase negative, and one variable strain, were used for injection into the caudal veins of groups of young mice. The lesions were observed histologically, quantitative determinations of the numbers of staphylococci in the organs at varying periods after infection were made, and survival times of the groups were recorded. All staphylococci disappeared rapidly from the circulation after injection, and apparently were destroyed, but a few living organisms persisted in the organs for several weeks. The kidney always proved to be the first site for the multiplication of both virulent and avirulent strains. The avirulent organisms produced abscesses which healed, while the virulent ones caused necrosis of the kidney tissue and death of the animal. The quantitative studies revealed that the avirulent strains, although increase did take place, never multiplied to the same extent as the virulent ones, and some of the latter multiplied to a greater extent than others, although all produced fatal infection. These test strains of staphylococci all failed to coagulate mouse plasma, although the virulence of the strain did appear to be correlated with coagulase production towards human and rabbit plasma.

Nutritional Disturbances and Staphylococcal Infections.

J. M. SMITH AND R. J. DUBOS (*J. Exper. Med.*, January, 1956) observed the effect of nutritional disturbances on the susceptibility of mice to staphylococcal infections. The strain of staphylococci used was the one which had been found to have the highest virulence in a previous series of experiments. Groups of mice were kept without food, but with ample water, for thirty-six hours; they were infected intravenously and were again fed. Controls were fasted, but not inoculated, and a third group was not fasted, but inoculated at the same time as the test group. There were variations apparent in the control group; individual mice differed in their susceptibility, but the fasted mice in each group had a lesser survival time than the properly fed ones. Quantitative determinations of the number of colonies showed larger numbers present in the fasted mice. The addition of glucose to the drinking water of fasting mice did not restore their resistance to infection; in fact it increased their susceptibility. A final group of mice were fed on a limited diet to prevent increase in body weight for several weeks. After inoculation by the intravenous route, this group behaved similarly to that of the well-fed mice.

Dinitrophenol and Thyroxin in Staphylococcal Infection.

J. M. SMITH AND R. J. DUBOS (*J. Exper. Med.*, January, 1956) have observed the effects of dinitrophenol and thyroxin on the susceptibility of mice to staphylococcal infections. Groups of mice were given drinking fluid containing either 1% sodium chloride, 0·01% dinitrophenol in a 1% solution of sodium chloride, or 0·03% thyroid extract in a 1% solution of sodium chloride. When these groups were infected with virulent staphylococci, the death rate was accelerated in those groups given dinitrophenol or thyroid extract, whereas uninfected control animals were retarded in growth, but did not die. When the animals were infected with avirulent staphylococci, deaths occurred in the treated mice, in contrast to the normal animals similarly infected. There was some suggestion that the thyroid treatment interfered with the bactericidal mechanism in the liver and spleen.

Enteric Virus Infection.

E. I. HONIG, J. L. MELNICK, P. ISAACSON, R. PARR, I. L. MYERS AND M. WALTON (*J. Exper. Med.*, February, 1956) have made an endemiological study of enteric virus infections, and have described poliomyelitis and orphan viruses isolated from normal children in two socio-economic groups. Orphan viruses are defined as strains of virus identifiable in tissue culture, non-pathogenic for monkeys and suckling mice, and not neutralized by poliomyelitis or Coxsackie antisera: "enteric cytopathogenic human orphan viruses ECHO". The authors studied children in normal households in non-epidemic periods; they used serum antibody measurements and virus isolation techniques, and maintained a careful record of minor illnesses. One group of children lived in poor homes, the other children were of good environments and were well nourished. Of 592 stool samples examined in the poorer district, 8·3% yielded virus, mainly in the summer months. In the better district with efficient sanitation, only 3·1% of stool samples carried virus. Monkey kidney tissue culture yielded 77 strains of virus, 44% of ECHO type, 37% of the Coxsackie type, and 19% poliomyelitis virus. Amongst the poliomyelitis virus carriers there were found antibodies to more than one virus type, but the homotypic antibody was persistent. Orphan viruses were present in more than the one child in a household; antibody to those isolated could be found in other members of the family.

Experimental Meningitis.

L. P. JERVEY, K. L. CLARK, D. G. FREIMAN AND M. HAMBURGER (*J. Infect. Dis.*, November-December, 1955) have continued their studies of experimental meningitis in rhesus monkeys. They used the strain of *Pseudomonas æruginosa*, previously established as a suitable agent to produce purulent exudate in monkeys. They infected the animals by the cisternal route, sacrificed them on the fifth or sixth day, and carefully collected and homogenized the tough yellow exudate. They then treated portions with solutions of streptokinase (this enzyme is known to activate plasminogen of human plasma to form plasmin), or streptodornase, a desoxyribonuclease which polymerizes extracellular DNA. These mixtures were mixed and placed in a water bath at 37° C. for four hours, and the turbidity was then estimated. The remaining exudate, after centrifugation, was washed, fixed in formalin, and stained by Feulgen's method. When the exudate was mixed with pure saline or serum saline, the supernatant remained clear. When enzyme mixtures were added, maximum turbidity of the supernatant appeared. These specimens, on staining of the centrifuged deposit, showed an absence or marked diminution of extracellular DNA. When the enzymes were used separately, streptokinase did not influence the amount of fibrin formation. An attempt was made to assess the minimum amount of dornase required to effect digestion, and something between two and twenty "units" of the preparations used were required over a period of four hours. Streptokinase, or a partially purified plasmin preparation, had no activity against the exudate, and no effect could be seen on the fibrin content. The same authors describe the effect of intracisternal injections of streptodornase or streptokinase on the exudate of experimental meningitis. Animals were infected as before, and cisternal punctures were performed daily. Fluid was obtained for analysis, and streptodornase alone or saline alone or a combination of the two was injected in a volume of one millilitre. The animals were sacrificed and the amount of exudate was assessed by an independent observer. The untreated animals had a larger amount of exudate than those treated by saline, while the animals treated by the enzyme had the least of all. When

large amounts of enzyme were injected, only one out of twelve animals displayed a large amount of exudate, compared with six out of eight untreated monkeys; the extracellular DNA was also reduced. Dornase activity persisted in the cerebro-spinal fluid for up to twenty-four hours, but no fibrinolytic activity could be demonstrated after the injection of streptokinase; the cause of this might have been in the absence of precursor in the fluid or instability under the conditions of the test. The authors believe that streptodornase might be a useful adjunct in the treatment of purulent meningitis.

Antibiotic Resistant Staphylococci.

V. G. ALDER, W. A. GILLESPIE AND M. M. E. THOMPSON (*J. Path. & Bact.*, October, 1955) have studied the virulence and phage patterns of antibiotic resistant staphylococci in a hospital. The cultures were taken from lesions in patients and nurses from 1953 to 1954; nasal swabs were examined at the same time, and settlement plates were exposed in the wards. Phage typing was carried out both by dilutions and by meat filtrates on otherwise untypable strains; 93% were typed. Of the cultures of *Staphylococcus pyogenes*, 421 were examined and 77·4% were found to be penicillin resistant, and most of them belonged to phage groups I and III. Group I strains were generally sensitive to all other antibiotics. Group III strains were less common than group I in the noses of nurses, and they were often resistant to more than one drug. Most of the furunculosis infections were associated with group I strains. There were very few group II strains isolated in this study.

HYGIENE.

Mercury Exposure in a University Laboratory.

L. GOLDWATER, M. KLEINFELD AND A. BERGER (*Arch. Indust. Health*, March, 1956) report the results of an investigation of the mercury hazard in a university laboratory in which mercury diffusion pumps were used. An assistant who performed a particular operation which resulted in an excessive exposure to mercury vapour developed signs and symptoms of mercury poisoning. Details of the operation and of the signs and symptoms are given. Other personnel employed in the same laboratory were studied, but showed no evidence of intoxication, because of their minimal exposure to mercury. Environmental studies revealed potential sources of danger which were amenable to simple control measures.

Service Statistics.

E. FLOOK (*Am. J. Pub. Health* February, 1956) defines service statistics in public health as numerical measurements of the services rendered to individuals and to the community through public health programmes. Useful service statistics should provide "operational data" that will help in the following ways: (i) furnish a basis for future public health programme planning; (ii) define and measure the extent of any public health problem; (iii) measure the service given and the progress made in relation to any such problem. Service statistics should reflect the service given in relation to base line data, previously established, relating to health problems to be solved. They should not merely enumerate the volume of staff activity, and they should be sufficiently limited both in scope and in volume to justify their production. The author considers that much work needs to be done in refining the significance of various units of measurement now being used and in refining and developing new and more useful units. When statistics, of the nature described, become an integral part of every basic public health design, the author considers that "we shall be in a much better position than we are today to tell where we are going in public health".

The Effect of Edathamil Calcium-Disodium on Lead Excretion.

R. BELL, J. GILLILAND, J. BOLAND AND B. SULLIVAN (*Arch. Indust. Health*, April, 1956) have investigated the urinary and fæcal excretion of lead before, during and after treatment by mouth or by the intravenous route with three grammes of edathamil calcium-disodium. It was found that when the compound was given by mouth, while men were out of exposure, there was a significant increase in both the fæcal and the urinary excretion of lead. The combined fæcal and urinary lead-excretion response, when the compound was given by mouth, was approximately two-thirds of the response obtained when edathamil calcium-disodium was given by the intravenous route. The increase in combined lead excretion with oral administration was approximately threefold. Increase was particularly marked in urinary lead excretion, especially with intravenous therapy. Data obtained suggested that fæcal lead excretion was diminished during intravenous therapy.

Some Effects of Diborane.

A. KUNKEL, E. MURTHA, A. OIKEMUS, D. STABILE, J. SAUNDERS AND J. WILLS (*Arch. Indust. Health*, April, 1956) have investigated the effect of diborane poisoning in animals. The use of boron hydrides in laboratories and industries is increasing. Less than one part per million is suggested as the maximum allowable air concentration for chronic exposure. It was found that the primary effect in poisoning by diborane appeared to be the production of pulmonary œdema. The earliest sign observed in dogs and rabbits during exposure was respiratory embarrassment. This was followed by a slight fall in blood pressure, an increase in activity of the smooth muscle of the intestine, and an increase and subsequent diminution in cortical activity. The latter effects were possibly associated with anoxia. Bradycardia occurred late in poisoning and was followed terminally either by ventricular fibrillation or by complete disappearance of ventricular activity.

School Lunch Rooms.

J. MCCUTCHEN (*Am. J. Pub. Health*, April, 1956) discusses the provision of hot meals in schools. As many of the facilities in use in schools were inadequate, the State Division of Health in Missouri drew up a scale of recommended requirements for school lunch rooms. Provision for the inspection of plans of all new installations was also made. Common defects in the plans inspected, together with recommended requirements, were as follows: (i) Inadequate space; the kitchen should have one and a half square feet for each meal served. (ii) Inadequate storage space; the storeroom should have one-half of a square foot for each meal served. (iii) Walk-in cooler not provided; schools serving 250 meals or more should have a walk-in cooler six feet by eight feet in area. (iv) Inadequate refrigeration; at least 20 cubic feet of refrigeration space should be provided for every 100 persons. (v) No clean dish racks; at least four racks for clean dishes should be provided. (vi) Dining area inadequate; there should be at least 10 square feet per person. (vii) Lack of hand washing facilities; one or more wash-hand basins with running hot and cold water should be provided. (viii) Failure to provide sneeze shields for cafeteria lines. (ix) Inadequate hot water facilities. Standards for hot water supplies, sinks and washing machines are discussed.

Development of the Present Concept of the Cause of Death.

I. MORIYAMA (*Am. J. Pub. Health*, April, 1956) refers to difficulties associated with the system now in use for the recording of causes of death. For public health purposes, the cause of death is the disease or injury that initiated the train of events leading to death. This concept was of value when public health was chiefly concerned with infective diseases. The increasing public health importance of chronic diseases has shown inadequacies in statistics now being compiled. The tabulation of multiple causes of death has been suggested, but little or no attention has been given to the uses and meanings of such tabulations. The author concludes that there is room for improvement in the quality of medical certification, the classification list, the classification procedures, the methods of presentation, and the uses of statistics on causes of death. However, he considers that once it is determined what information such statistics should supply, steps can be taken to alter one or more of the following in order to evolve a system that will supply the desired information: the basic medical information to be supplied by the physician; the method of reporting; the classification list of diseases; and the method of selection of causes to be tabulated.

Testing of Frozen Fish Products.

E. LARKIN, W. LITSKY AND J. FULLER (*Am. J. Pub. Health*, April, 1956) draw attention to the increased consumption of frozen fish products and to the need for clean processing and handling. The numbers of coliform bacteria and fæcal streptococci in samples of frozen fish and fish products are compared. They advocate the use of fæcal streptococci as test organisms for the bacteriological examination of frozen fish products. The testing procedures for the isolation of fæcal streptococci are described. The authors consider these tests to be more simple and the results to be of more significance than those for the coliform bacteria. For accurate results, a blendor is used to mix the fish with sterile tap water. The resulting mixture is used for testing. Results sufficiently accurate for public health supervision can be obtained by shaking the products in water by hand. Plate counts were found to be of little value.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at Prince Henry's Hospital, Melbourne, on March 21, 1956. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

Miliary Tuberculosis.

DR. KEITH BOTTOMLEY presented a girl, aged nineteen years, who had been suffering from miliary tuberculosis. She had been admitted to hospital with a history of a few days' pyrexial illness, shortness of breath being the main symptom. Her temperature was 104° F. on admission to hospital and she had orthopnœa and generalized bronchitic sounds throughout both lungs. X-ray examination showed miliary lesions throughout both lungs. The sputum contained *Mycobacterium tuberculosis*. This organism was also grown from the urine, and X-ray examination of the renal tract showed double ureters on both kidneys. Tubercles were seen in the choroid, and there was electrocardiographic evidence of ventricular damage. The patient was treated with routine doses of streptomycin and "Isoniazid", and after four months' treatment was symptomless and afebrile, and the X-ray appearance of the chest and electrocardiogram were normal. She still had a raised erythrocyte sedimentation rate. No primary lesion was discovered.

Lymphosarcoma.

Dr. Bottomley's second patient, a man, aged fifty-three years, had been admitted to hospital with a seven-months history of glandular enlargement in neck and axilla. On examination he was found to have generalized discrete lymph gland enlargement. Examination of an axillary lymph follicle showed changes typical of a giant follicular lymphoma. Examination of a gland removed from the opposite axilla showed sarcomatous degeneration in addition to the lymphomatous change. He was treated with "Colcemid" (demecolcine, an antimitotic extract from colchicine) with a decrease in the size of the lymph glands. The dosage of "Colcemid" had to be reduced owing to the fall in the number of white cells to 300 per cubic millimetre. Over the past month the glands had gradually increased in size, and no further benefit had been obtained from "Colcemid" therapy.

Spontaneous Pneumothorax (Possibly Tuberculous).

DR. THEO FRANK showed a male patient, a recent Greek migrant, aged thirty years, who had been admitted to Prince Henry's Hospital on February 20, 1956, with a history of acute chest pain of dramatically sudden onset, associated with coughing and severe dyspnœa. The pain was severe, sharp in nature and situated in the left pectoral region from the third to the eighth rib levels, extending posteriorly. The classical signs of a left pneumothorax were present, and the temperature was 98·8° F. The blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. There was no past history of pneumothorax or of association with tuberculosis. A diagnosis of left spontaneous pneumothorax was made, and treatment was commenced with 600,000 units of penicillin and half a gramme of streptomycin twice daily and analgesics.

Pathological investigations at this stage revealed a normal blood film and normal flora in the sputum. Repeated sputum tests for tubercle bacilli yielded negative results. The Mantoux reaction was strongly positive to 1:1000 old tuberculin on February 22 and 27, 1956, after forty-eight hours. Serial X-ray examinations showed progressive reexpansion of the upper lobe of the left lung and early signs of pleural effusion.

On February 27, 1956, the patient developed chest pain, and the temperature, which had reverted to normal, rose to 101° F. On March 2 there was a gradual onset of severe pleuritic pain, which over the next few hours increased and caused distress. The temperature rose to 102° F. A check X-ray examination on this day revealed complete collapse of the left lung, and fluid had accumulated to the tenth intercostal space. A diagnostic pleural aspiration yielded yellow opalescent fluid, containing numerous polymorphonuclear cells, 4·9 grammes *per centum* of protein and no bacteria. Culture for tubercle bacilli was in progress. About twelve hours after the onset of the recurrent pneumothorax, 1000 cubic centimetres of air were removed with a specially designed pneumothorax machine. A further X-ray examination on the next day showed 80% reexpansion of the lung, but still considerable fluid at the base of the left lung.

The following regime of therapy was instituted: penicillin 500,000 units four-hourly, streptomycin half a gramme twice daily, "Isoniazid" 300 milligrammes daily. On March 7, 18 ounces of straw-coloured fluid were aspirated, and three days later a further 36 ounces. At the present time the temperature pattern was normal, and the patient subjectively well. A check X-ray examination made on March 13, 1956, showed that the lung had almost completely reexpanded, and only a minimal amount of fluid was present.

Dr. Frank commented that most cases of complete spontaneous pneumothorax were due to rupture of an emphysemetous bulla. However, the cause in the present case was probably tuberculous, the features in favour of that diagnosis being a very strongly positive response in the tuberculin test, the elevated temperature and the development of an effusion with a high protein content. Until the diagnosis was established, antituberculosis treatment would be given.

Aneurysm of Internal Carotid Artery: Tuberculous Lymphadenitis.

Dr. Frank next showed a married woman, aged fifty-six years, who had been first examined by him on August 31, 1950, when she gave a history of severe headaches for three years, associated with nausea. (X-ray examination of the skull and sinuses in 1947 had shown no abnormality.) She had also had double vision for six months on looking to the right, and dyspnœa on effort. Her past history was clear. On examination of the patient, her blood pressure was 220 millimetres of mercury, systolic, and 120 millimetres, diastolic. She had also a loud aortic second sound and paralysis of the right fourth and sixth cranial nerves. Examination of the fundi showed retinal arteriosclerosis. The result of a blood Wassermann test was negative.

X-ray examination of the skull on November 15, 1950, showed erosion of the anterior clinoid process on the right side. The *dorsum sellæ* was thinned, and the floor of the pituitary fossa had a double outline most probably due to some enlargement on the right side. The margin of the right optic foramen and the right sphenoidal ridge was not quite as well defined as that on the left side. The cerebral falx was calcified and was displaced slightly to the left, but the pineal gland, which was also calcified, was in the mid-line. Cerebral angiography showed a large aneurysm one and a quarter inches in diameter rising from the internal carotid artery in the region of the pituitary fossa; the anterior cerebral artery was poorly developed.

On November 30, 1950, ligation of the right common carotid artery was performed. On the third day after operation the patient developed complete right third nerve palsy and loss of sensation of the first and second divisions of the right fifth nerve (probably due to shrinkage of the aneurysm causing traction on those nerves). On December 13, 1951, the headaches had disappeared, the third and fourth nerves had largely recovered and the fifth nerve was normal. The patient still had diplopia in certain positions.

On December 13, 1955, the patient felt fit. She still had diplopia in certain positions and occasionally had nervous headaches. "Lumps" had been present in the left supraclavicular triangle for two months. The blood pressure was 220 millimetres of mercury, systolic, and 140 millimetres, diastolic. In the left supraclavicular fossa there was a mass of glands above the clavicle with smaller discrete glands above that mass. The liver and spleen were not felt. No other glands were palpable. X-ray examination of the chest showed an old calcified focus in the left infraclavicular region. No mediastinal glands were visible, but there was atherosclerotic dilatation of the aorta. The results of blood examination were normal. The Mantoux response was positive at a dilution of 1:1000 after forty-eight hours. On examination of the sputum no tubercle bacilli were found; a specimen was sent for culture. X-ray examination of the sinuses showed no abnormality and the X-ray appearances of the cervico-dorsal part of the spine were apparently normal. Examination of the biopsy specimen showed that it contained firm matted lymph nodes, which on section were seen to contain large central areas of caseation surrounded by tuberculous lymphadenitis.

Dr. Frank said that the interest in the patient was twofold: (i) An aneurysm of the internal carotid artery was responsible for severe headaches and double vision. Ligation of the common carotid artery gave her relief from her symptoms of intolerable headaches and also prevented further

enlargement of the aneurysm with fatal rupture. (ii) The occurrence five years later of a mass of tuberculous glands in the left supraclavicular fossa was difficult to explain. Although there was a healed tuberculous lesion in the left supraclavicular region (as shown by X-ray examination), a spread from there to the neck seemed unlikely.

Treatment had consisted of streptomycin one gramme given intramuscularly three times weekly and "Isoniazid" 300 milligrammes daily. As a result, the glands had rapidly decreased in size, and the biopsy wound had healed quickly. It was proposed to continue the treatment for at least six months and then to review the progress. At the present time it seemed very unlikely that surgical excision of the glands would become necessary.

Possible Hypogonadism or Frölich's Syndrome.

Dr. Frank's third patient was a boy who had been first examined on August 3, 1945, when he was aged fifteen years. He then had strained neck muscles and a bruised right buttock, the result of a fall. His voice was high-pitched, and there was an absence of hair on the face, in the axillæ and above the *symphysis pubis*. The sexual development was poor. There was excess fat on the body, chest and pubic region. His height was five feet eight and a quarter inches and his weight twelve stone eleven and a half pounds. He was referred to the Royal Melbourne Hospital out-patient department for treatment with a 1200-Calorie diet and thyroid, and was also given a course of "Antuitrin S" injections weekly for six weeks. As a result he lost about two stone in weight.

The following results were obtained from investigations carried out in July, 1951: glucose tolerance curve, flat type; serum cholesterol content, 195 milligrammes per 100 millilitres; basal metabolic rate, −26%; X-ray appearances of the pituitary fossa, normal; visual fields, normal. An encephalogram revealed a faint shadow projecting upwards from the pituitary fossa in the chiasmatic cistern. It probably represented a suprasellar extension of some pituitary tumour. No operative interference was indicated.

On May 8, 1954, he reported for treatment of constipation. His weight was ten stone eleven and a half pounds and his height five feet nine and a quarter inches. His voice was still high-pitched, and there was no growth of hair on his face, forearms and armpits, with very little hair on the *mons pubis*. The penis and testicles were small.

Treatment was started on August 21, 1954, with testosterone 100 milligrammes given intramuscularly every fourteen days. On December 18 his voice was deeper, and there was more hair above the *symphysis pubis*. On February 19 his voice was still deeper, whilst the eyebrows and axillary hair were growing. On April 30, 1955, his penis had shown considerable enlargement, and his testicles had increased in size. Occasional erections and seminal emissions occurred. On February 16, 1956, he had no hair on the face or forearms, but a small amount on the upper lip. There was hair in the armpits and also a good growth above the *symphysis pubis* with an extension towards the umbilicus. The testes were still small, whilst the breasts were still enlarged. His weight was ten stone thirteen and a half pounds.

Dr. Frank said that the patient had been examined by Professor Astwood at a clinical demonstration in 1954, and he considered the condition to be primary hypogonadism despite the findings suggestive of a pituitary tumour in an encephalogram in 1951. After androgen therapy, there had been a great improvement in the patient's general health and also in the development of secondary sexual characteristics. However, there was still a lack of facial hair growth, whilst the testicles were still below normal size and gynæcomastia was present.

Myelofibrosis.

Dr. Frank's last patient was a single woman, aged sixty-four years, formerly a school teacher, who had been examined on February 19, 1951. She had then a history of chilblains on the backs of the legs in winter time for years past. An area of cellulitis on the left shin and later on the right shin had developed in December, 1950; it had responded quickly to penicillin injections and rest, but had recurred three days before her coming for the present consultation. Her general health had been good. She had three sisters and one brother, all alive and well. Her father had died at the age of seventy-five years of coronary disease, and her mother at the age of eighty-three years of leuchæmia.

Examination of the patient showed splenic enlargement four fingers' breadth below the left costal margin, and thickening of the skin of the lower third of both legs. There was no glandular enlargement. Piles were present. A blood count on February 21, 1951, showed a total of 3,300,000 erythrocytes per cubic millimetre, a hæmoglobin value of 70% (9·9 grammes *per centum*) and a colour index of 1·03. The leucocytes totalled 5700 per cubic millimetre, made up of polymorphonuclear cells 47%, old metamyelocytes 15%, young metamyelocytes 4·5%, myelocytes 7%, lymphocytes 15·5%, eosinophile cells 8% and basophile cells 3%. The red cells were well hæmoglobinized and showed a fair amount of anisocytosis. Their size varied from macrocytic to microcytic, but there were many normal in size. Platelets were present. Three erythroblasts were seen during the counting of 200 white cells. Reticulocytes appeared to be under 1% of the total red cells, and there was occasional polychromasia.

On March 5, 1951, examination of bone marrow showed a slight increase in neutrophile cells, but otherwise a normal distribution. There was no evidence of leuchæmia. The findings of barium meal examination and excretion pyelography were normal. A blood count on August 2, 1951, showed a hæmoglobin value of 59%, an erythrocyte count of 3,050,000 per cubic millimetre, and a colour index of 0·98. The leucocytes numbered 7600 per cubic millimetre with a differential count similar to that obtained on February 21, 1951. Reticulocytes made up 3·5% of red cells. Diffusely polychromatic cells were fairly numerous. Two normoblasts were seen during the counting of 200 leucocytes. The red cell fragility was increased, hæmolysis beginning at between 0·56% and 0·60% strengths of saline and being complete at 0·36%. The result of a Coombs test was negative. Urobilin in the urine was in very small excess. Stercobilin in the fæces amounted to 30 milligrammes per 100 millilitres. X-ray examination of the long bones showed a little patchy relative rarefaction in the upper end of the right humerus, with a similar though less pronounced appearance on the left side. Sternal marrow biopsy on June 19, 1953, showed that there were no marrow elements in the intertrabecular spaces of the bone, which were occupied by an œdematous tissue poor in fibrils. A little dense fibrous tissue was seen. The findings were considered to be suggestive of myelofibrosis.

Late in June, 1955, the patient complained of dizzy turns, pains in the legs and great weakness. Examination showed that the spleen had enlarged into the left iliac fossa, and that the liver was palpable several fingers' breadth below the right costal margin. The hæmoglobin value was now 50% (6·5 grammes *per centum*). A further blood count on July 21, 1955, showed a hæmoglobin value of 31% (4·6 grammes *per centum*), a hæmatocrit of 15%, a white cell count of 12,000 per cubic millimetre, and 7·0% of reticulocytes. Examination of the blood film showed extensive anisocytosis with marked microcytosis and poikilocytosis (mainly small oval cells), slight macrocytosis and polychromasia and slight hypochromia of a few microcytes. The platelets were probably somewhat reduced. An occasional polymorphonuclear cell was macrocytic and multisegmented. Both polymorphonuclear and eosinophile cells showed toxic granulations in some cells. During the counting of 100 white cells, five nucleated red blood cells were seen. In a differential count of the white cells there were myelocytes 14%, metamyelocytes 10%, staff forms 5%, segmented neutrophile cells 38%, eosinophile cells 8%, basophile cells 2%, lymphocytes 20% and monocytes 3%.

The patient was admitted to Prince Henry's Hospital on August 3 and given two pints of blood and two pints of packed red cells; as a result her hæmoglobin value rose to 80% (11·6 grammes *per centum*). On September 20 she was readmitted to hospital with the following blood picture: hæmoglobin value 18% (2·6 grammes *per centum*), red cell count 1,160,000 per cubic millimetre, white cell count 3900 per cubic millimetre. She was given a transfusion of four pints of packed cells and two pints of whole blood. On October 4 her hæmoglobin value was 72% (10·4 grammes *per centum*). Since then her health had been good, and on February 14 her hæmoglobin value was 66% (9·6 grammes *per centum*).

Dr. Frank summed up the case as one of myelofibrosis in a patient under observation for five years, with maintenance of fair health apart from two episodes in August and September, 1955, when there was a marked increase in anæmia for no apparent reason; blood transfusions helped her over those two attacks. Dr. Frank said that the use of cortisone had been considered if there had been a recurrence of severe anæmia. The question of splenectomy had been raised, and it might have been used as a final resort if there had been evidence of destruction of red cells by the spleen. In view of the widespread involvement of the

reticulo-endothelial system in myelofibrosis, splenectomy was, as a rule, contraindicated.

Hæmochromatosis.

DR. A. SEWELL showed a patient suffering from hæmochromatosis, who had received 180 venesections in the past eighteen months, averaging fifteen ounces each time. The improvement in his condition had been dramatic. His hepatic enlargement, which had been extremely gross, was now only one finger's breadth below the costal margin. The blood sugar curve, which had been frankly diabetic, had reverted to a normal curve, and the serum iron level had fallen from 250 milligrammes to 50 milligrammes per 100 millilitres. The patient was symptomatically well and had entirely lost his most distressing symptom—namely, severe muscle cramps.

Addisonian Crisis following a Mantoux Test.

Dr. Sewell's second patient had been examined eighteen months previously, when he had an attack of pleurisy. After subsidence of the pleurisy, a Mantoux test was carried out with 1:1000 old tuberculin. This was followed by a general febrile reaction, a large local reaction at the site of injection and a return of pleural pain with reappearance of the friction rub. Fifty-six hours after the test, acute collapse developed with severe dehydration, fall of blood pressure to 60 millimetres of mercury, systolic, and 20 millimetres, diastolic, and a fall of blood sodium level to 265 milligrammes per 100 millilitres. The crisis was controlled by intravenous administration of saline and cortisone, but Addison's disease had persisted and cortisone was still required for control of the disease. This patient was presented as showing an interesting sequel to a Mantoux test.

Long-Standing Exophthalmic Ophthalmoplegia.

The last patient shown by Dr. Sewell had suffered from severe exophthalmus with exophthalmic ophthalmoplegia for five years. She was not thyrotoxic. Four millilitres of hyaluronidase were injected subconjunctivally into each eye, and within twenty minutes the ophthalmoplegia had disappeared. The improvement had been maintained over a period of three months.

Scleroderma.

DR. H. W. GARLICK showed two patients suffering from scleroderma.

The first was a woman, aged fifty-two years, who in October, 1955, had complained that she had stiffness in both shoulders on waking in the morning and swelling and stiffness of the fingers. On cold days the tips of the fingers would be blue, and at times the distal half of a finger would go white. In bed at night the tips of the fingers would throb; that was worse when the hands were warm, and for that reason she slept with her hands on top of the bedclothes.

On examination of the patient at that time the only abnormalities found were that the fingers were cold and swollen, and she could not clench her fist tightly, and that the skin over the forehead and front of the neck seemed tight. Skin biopsy from the neck provided confirmatory evidence for the diagnosis of scleroderma.

Dr. Garlick said that she had been treated with "Atebrin" 100 milligrammes daily, and she had been much better (the weather had not been cold).

The second patient was a woman, aged forty-nine years, whose illness had begun in 1943 with loss of energy and weight, and pain, stiffness and swelling of various joints, but particularly of the small joints of the hands. At that time she was diagnosed as suffering from atypical rheumatoid arthritis. Over the next few years there was a gradual stiffening and contracture of the fingers and toes. In 1948 she noticed that her mouth seemed small, and the skin of her face tight. In the winter her hands were cold and painful with ulceration of the finger tips. There had been no dysphagia.

Disseminated Lupus Erythematosus.

Dr. Garlick then presented details of three patients suffering from disseminated *lupus erythematosus*.

The first was a woman, aged twenty-seven years, who in 1951 had been sunburnt. After that she developed a rash on the face which was diagnosed as *lupus erythematosus*. From 1951 to 1953 she was treated intermittently for skin lesions. In March, 1954, she was admitted to hospital with high fever, loss of weight and energy, and vomiting. On examination she was found to have a temperature of 103° to 104° F., extensive skin lesions of lupus, albuminuria and *lupus erythematosus* cells in the blood. She made a dramatic response to cortisone, but heavy albuminuria persisted. Apart from several intercurrent infections she was in reasonable health until December, 1955. Cortisone therapy was continuous. She died on February 28, 1956, with renal failure and bleeding from the nose and gastro-intestinal tract.

Dr. Garlick said that it was well recognized that cortisone had no effect on the renal lesions in systemic *lupus erythematosus*. The histology of the renal lesions in disseminated *lupus erythematosus* was well shown in sections presented at the meeting.

The second patient was a girl, aged nineteen years. It was stated that in January, 1955, she had been sunburnt on the face, arms and legs; she responded to treatment with a lotion. Two weeks later she became "sunburnt on the face" although she was not exposed to the sun. Her doctor told her to stop drinking tea and prescribed "sulpha" tablets. Over the next six weeks she felt generally off colour with no appetite and no energy. The fingers and knees were swollen, stiff and at times painful. Loss of weight (eleven pounds) occurred. The rash on the face persisted, and after a few weeks spread to the shoulders, extensor aspect of the arms and medial aspect of the thighs and legs. Her past history was that she had had scarlet fever, at the age of four years, and "rheumatism" at the age of fourteen years, when she was in bed for three months.

In March, 1955, she was admitted to hospital. The following abnormal findings were recorded at that time: A loud blowing apical systolic murmur was audible. The fingers of both hands were swollen. Red scaly patches were present across the bat's wing area of the face, shoulders and dorsal aspect of the arms. Dermatological opinion at that time was that the rash was due to seborrhœic dermatitis. For a week or so the temperature was up to 99·4° F. only, but suddenly it went up to 103° to 104° F. Neutropenia was present. *Lupus erythematosus* cells were found in the blood. There was a dramatic response to cortisone, and that was continued for three months. She had remained well since.

The last patient was a woman, aged twenty-eight years, whose illness had begun in February, 1955, with pain, swelling and stiffness in the wrists, small joints of the hands, shoulders and later knees. The diagnosis was considered to be rheumatoid arthritis. On May 27, 1955, she developed a sore throat, cough, high fever and delirium. On May 28, 1955, she had bilateral deafness. On June 1, 1955, her speech was slurred, and the right ear started to discharge a watery fluid. She was admitted to hospital with a provisional diagnosis of intracranial abscess. The following abnormal findings were recorded on admission to hospital: high fever (103° to 104° F.); mental confusion; nystagmus; left plantar reflex extensor; ear drums—right perforated, left bulging; joint changes compatible with rheumatoid arthritis. Her cerebro-spinal fluid was normal. Attempted blood cultures produced negative results. Left myringotomy produced serous fluid from which *Staphylococcus aureus* was grown. Persistent neutropenia was present. X-ray examination of skull and chest showed no abnormality. There was no response to antibiotic therapy. Numerous *lupus erythematosus* cells were eventually found in the blood, and on June 13, 1955, cortisone therapy was commenced. The response to cortisone was dramatic, and on July 5, 1955, she was discharged to her home receiving cortisone 25 milligrammes three times a day. Dr. Garlick said that over the last eight months she had had several attacks of "pneumonia", which her doctor had found responded to penicillin and no other antibiotic. In addition he had found it necessary to increase the dose of cortisone gradually up to 225 milligrammes a day. She was readmitted to hospital in March, 1956, and now showed many of the features of Cushing's syndrome in addition to features of disseminated *lupus erythematosus*. Her therapy had been changed from cortisone to "Delta Cortef".

Cardiac Murmurs.

DR. ALLAN WYNN presented a series of patients to illustrate various kinds of cardiac murmurs.

Sarcoidosis in Identical Twins.

DR. IAN JONES showed two patients, who provided an example of sarcoidosis occurring in identical twins.

The first patient, a married woman, aged twenty-seven years, had been first examined by Dr. Jones on December 21,

1954. She then gave the following history. In 1948 she had developed lumps on both legs associated with uveitis and enlargement of lymph glands, including hilar glands. Dyspnœa on exertion was noticed at that time. The episode lasted for six months and settled spontaneously. Three years later she presented with generalized lymphadenopathy, dyspnœa and skin rash. She was investigated elsewhere, X-ray examination showing enlarged hilar lymph glands, and biopsy of inguinal glands showing a "typical histology of Boeck's sarcoid". Over the subsequent three years, intermittent lymph gland enlargement had occurred, and the skin rash had persisted. In 1952 an attack of hæmaturia had occurred, and for three months she had noticed increasing lethargy, anorexia, insomnia and pain associated with swelling in the left hypochondrium. Pruritus, particularly of the face and hands, irritating cough and dyspnœa had been present for two months. On examination of the patient a large tender spleen was felt extending to the umbilicus. Enlargement of the upper cervical and left axillary lymph glands was moderate. The skin of the thighs, shins and head showed discrete, irregular, retracted pigmented areas. The biopsy scar showed similar pigmentation. There was no evidence of uveitis or choroiditis, and the lungs were clinically clear. X-ray examination of the chest showed fine, diffuse, nodular shadowing of both lungs without hilar gland enlargement. Blood examination showed a hæmoglobin value of 11·6 grammes *per centum* and a leucocyte count of 3600 per cubic millimetre with a normal differential count. Platelets numbered 129,000 per cubic millimetre. Bleeding and clotting times were normal. The result of a Coombs test was negative. The findings from excretion pyelography and urine examination were normal. The total serum protein content was 8·9 grammes *per centum* (albumin 4·1 grammes, globulin 4·8 grammes *per centum*, albumin/globulin ratio 1:1·2). Later her total serum protein content rose to 10 grammes *per centum*, with a similar ratio to that on the previous occasion. The results of Mantoux tests were negative with dilutions of 1:1000 and 1:100 old tuberculin. The erythrocyte sedimentation rate was 42 millimetres in 60 minutes (Wintrobe). The result of a cephalin flocculation test was positive. Bone marrow biopsy showed increased normoblastic erythropoiesis; no abnormal cells were seen. X-ray examinations of hands, femora and pelvis showed no abnormality. The results of Wassermann and Kahn tests were negative.

Owing to persisting and often disabling pain at the site of a greatly enlarged spleen, splenectomy was performed on March 16, 1955. The splenic substance was largely replaced by tubercle-like follicles comprised mainly of multinucleated giant cells and epithelioid cells. No caseation necrosis was seen. "Asteroid" inclusions were present in many giant cells. The histological picture was typical of sarcoidosis throughout the spleen. The results of animal inoculation and culture for tubercle bacilli from the spleen were negative.

Convalescence was uneventful, and with no other treatment (other than symptomatic for minor ailments) the patient subsequently enjoyed good health. Serial chest X-ray films showed no significant change. The last blood examination on January 6, 1956, had shown a hæmoglobin value of 16·8 grammes *per centum*, a leucocyte count of 4500 per cubic millimetre and an erythrocyte sedimentation rate of eight millimetres in one hour.

The second patient, also a married woman, an identical twin of the first, was interviewed while visiting her sister in hospital. Although feeling well, she consented to physical examination and limited investigation.

On direct questioning on March 25, 1955, she admitted to tiring easily, some dyspnœa on exertion and tightness in the throat on eating at times. Like her twin sister, she had been troubled with varicose veins, had had menstrual upsets and had borne no children. On examination of this patient the spleen was definitely palpable, and the thyroid gland was slightly enlarged, but no other definite abnormality could be detected. In particular there were no palpable glands. X-ray examination of the chest showed a moderate increase in hilar shadows presumably due to lymph gland enlargement. The lung fields were clear. Full blood examination showed a hæmoglobin value of 13·9 grammes *per centum*, a leucocyte count of 3400 per cubic millimetre with a normal differential count, and 167,000 platelets per cubic millimetre. The results of Mantoux tests were negative with dilutions of 1:100 and 1:1000 old tuberculin. The total serum protein content was 6·8 grammes *per centum* (albumin 4·8 grammes, globulin 2·0 grammes *per centum*, albumin/globulin ratio 2·4:1). The erythrocyte sedimentation rate was four millimetres in sixty minutes (Wintrobe).

On August 19, 1955, a laparotomy was performed for acute abdominal pain, and an ovarian cyst and the appendix were removed. Examination of a section of the former showed simple cyst and ovarian tissue only.

Although it was not proven, it was felt that the association of hilar gland enlargement and splenomegaly in an otherwise healthy Mantoux-negative person was at least suggestive of sarcoidosis. No enlarged lymph glands had been felt suitable for biopsy, and it had not been considered justifiable, as yet, to proceed with liver biopsy. Arrangements had been made for a Kviem test to be performed on each patient, as it was thought that a positive response in the case of the second patient would strengthen the diagnosis.

Pyrexia of Uncertain Origin.

Dr. Jones then discussed two unusual cases of pyrexia of uncertain origin.

In the first case, that of a man, aged fifty-seven years, the condition was still undiagnosed. The illness had commenced in January, 1952, with lassitude and somnolence. Pyrexia was first discovered in December, 1952, and had continued ever since, except when the patient was receiving full dosage of cortisone. Frequent frontal headaches and an occasional feeling of faintness were also complained of, but otherwise he was free of symptoms. He was first admitted to hospital for investigation on March 19, 1953. Physical examination revealed no abnormality other than pyrexia. The only positive findings in an exhaustive investigation were the following. Full blood examination showed mild anæmia (a hæmoglobin value of 10·3 to 13·0 grammes *per centum* and a colour index of 0·93) with mild inconstant neutrophilia; the erythrocyte sedimentation rate was 54 millimetres in sixty minutes (Wintrobe). Chest X-ray examination was repeated many times in view of the early presence of left basal atelectasis; subsequent films while the patient was still febrile were clear. Ventriculography showed slight dilatation of the lateral ventricles suggestive of a moderate degree of cerebral atrophy. The result of a Mantoux test was positive with a dilution of 1:100 old tuberculin. Various liver function tests were carried out: the serum alkaline phosphatase content was 22·4 King-Armstrong units, the result of the cephalin flocculation test was positive, and other findings were normal.

Numerous antibiotics were given, and he was discharged to his home still febrile, having lost two stone in weight, on August 8, 1953. At home his condition worsened, and he was readmitted to hospital on September 29, 1953, for six months. He was somnolent and had two to three fingers' breadth of liver enlargement. On the right calf there was a macular rash covering a small area.

On October 14, 1953, an exploratory laparotomy was performed and showed the liver slightly enlarged and smooth, and slight splenomegaly, but no other abnormality. Biopsy of the liver (a previous blind needle biopsy had been unsuccessful) showed a focal granulomatous lesion of the liver associated with fibrosis and fatty change. Examination of the focal granulomata showed necrosis of liver cells, accumulation of small mononuclear cells predominantly, and some polymorphonuclear and eosinophile cells. There was evidence of fibrosis around the granulomata and throughout their substance. The portal tracts showed increase in collagenous tissue, and there was a definite thickening of the reticular framework of the lobules. The granulomatous lesions were not characteristic of any specific infection.

The patient became afebrile within forty-eight hours of the start of treatment with cortisone, 100 milligrammes three times a day, on December 23, 1953. The dose was gradually reduced; but if it was dropped below 37·5 milligrammes three times a day, fever would return. He was discharged from hospital on this dose. The rapid sedimentation rate dropped to normal within three weeks of the institution of cortisone treatment.

Cortisone dosage was continued, but owing to increasing difficulty in controlling pyrexia, he was readmitted to hospital on March 16, 1955, for further liver biopsy. Three fingers' breadth of liver enlargement was present, and the spleen was palpable. Liver biopsy on March 21, 1955, showed appearances similar to but less intense than those seen in the previous biopsy. There was no evidence of progressive fibrosis. Again an unsuccessful attempt was made to grow organisms from the specimen. The result of a cephalin flocculation test was then negative, but the alkaline phosphatase content was still raised (27 King-Armstrong units). The serum proteins remained normal in amount.

Since his discharge from hospital on March 27, 1955, the patient had remained reasonably well and able to work,

apart from an acute perianal abscess (due to *Staphylococcus aureus*) in September, 1955. However, if the dose of cortisone was reduced below 25 milligrammes three times a day, his temperature rose.

Dr. James said that it was unlikely that an infection could behave in such a manner over so many years in the absence of any systemic localization. A diagnosis of sarcoidosis was suggested, but it would indeed represent an odd clinical course. Such a diagnosis was not supported by liver biopsy. The same applied to systemic *lupus erythematosus*. A search for *lupus erythematosus* cells had been unsuccessful. Another possibility under consideration was a neurogenic fever. Could there be some obscure lesion of the hypothalamus? Dr. R. Doig had suggested that there might be a neurogenic basis to the pyrexia similar to that seen in the periodic syndrome in children. The patient was meticulously "finicky", having kept a detailed diary of his symptoms throughout his illness.

The second case, that of a married woman, aged thirty-five years, illustrated an unusual mode of presentation of bronchial adenoma. For seven years she had suffered from recurrent bouts of fever characterized by rigors, generalized aches and pains in the right side of the chest, and sore throat. She remembered fifteen attacks in all, subsiding spontaneously within seven days. Her chest had been examined radiologically several times, and the findings had always been normal. No X-ray film had been taken at the height of an attack.

In one such attack she was admitted to hospital on August 28, 1954. This episode differed from the others in duration only, having already been present for eight days. X-ray examination of the chest revealed collapse of the middle lobe of the right lung, with some pneumonic consolidation of the base of the upper lobe of the right lung. Bronchoscopic examination revealed a benign-looking tumour occluding the middle lobe bronchus. A right middle lobectomy was performed by Dr. Hamley Wilson on November 18, 1954. Examination of the specimen showed a large adenoma completely occluding the distended middle lobe bronchus. The adenoma measured 1·8 by 1·0 by 1·0 centimetres. The distal bronchi were moderately dilated, and the lung tissue showed fibrosis and intense chronic inflammatory reaction.

After a stormy convalescence, the patient had remained well. It seemed probable that the bouts of fever were due to recurrent infection in the right middle lobe, complicating intermittent obstruction by a bronchial adenoma. There was no history of hæmoptysis, and at no time was cough a prominent symptom.

Anatomy and Technique of Appendicectomy.

DR. DOUGLAS DONALD presented a demonstration designed to help to improve the technique of appendicectomy by showing the anatomy of the area (the anterior abdominal wall and the alimentary tract in the vicinity of the appendix). The demonstration revealed the following points: (i) The skin incision should be placed more laterally. (ii) The internal oblique and transversus muscles might be split (*a*) with minimum damage of those muscles and (*b*) without pressure on the abdominal contents, so that risk was avoided of disturbing any underlying infection or abscess. (iii) The site of the appendix was such that the operation need not be delayed more than three minutes after opening of the peritoneum in finding the appendix. (iv) The site of the base of the appendix was constant. (v) Adhesions of the body of the appendix were very rare, apart from the appendix involved in an abscess. Jackson's membrane was the structure so often referred to as "adhesions". The clearing of that membrane was a minor point in the technique of appendicectomy.

Lymphœdema Præcox.

DR. S. F. REID showed a woman, aged twenty-one years, with *lymphœdema præcox*. The onset of swelling had occurred at puberty and had been fairly gross for two years. Symptoms were cosmetic *plus* considerable aching in the calves and feet. The swelling extended from the toes to above the knee and was of considerable degree. The patient was given various hormones, orally administered diuretics and ion exchange resins, all without effect. Remarkable and sustained improvement followed injection of "Mersalyl" two millilitres twice per week for five weeks. There was no recurrence of swelling in the subsequent four months over the hottest period. Dr. Reid's comment on the case was that it illustrated an unusual and very satisfactory response to mercurial diuretics by injection in *lymphœdema præcox*.

Carpal Tunnel Syndrome.

The second patient, a woman, aged twenty-six years, had carpal tunnel syndrome of the right hand; with typical nocturnal paræsthesia, wasting and weakness of thenar muscles, symptoms of *tenovaginitis stenosans* in the tunnel and pronounced defects upon use of the long flexors of the fingers. A large lump was present proximal to the flexor retinaculum in the mid-line of the distal part of the forearm and a similar lump in the proximal hollow of the palm. The mass was semifluctuant and non-translucent, and crepitus was detectable over it just proximal to the flexor retinaculum. Clinically it resembled a compound palmar ganglion, but there was no fluid extension up the ulnar bursa to the little finger. The results of a Mantoux test and X-ray examination of the chest were negative. At operation exploration and transection of the flexor retinaculum were carried out. Biopsy of material from the sheath and nerve showed œdematous inflammatory tissues. Complete relief of symptoms followed operation, with recovery of the short muscles of the thumb and disappearance of anæsthesia. The patient since had minor symptoms in the other hand completely relieved by small doses of stilbœstrol given by mouth. Dr. Reid said that this was an unusual case of carpal tunnel syndrome with a large lump which was almost entirely median nerve and caused confusion in diagnosis with compound palmar ganglion. Attention was drawn to the fact that many cases of compound palmar ganglion clinically showed no extension of fluid up the ulnar bursa, and it was probable that many of those were misnamed and erroneously regarded as tuberculous.

Traumatic Rupture of the Diaphragm.

The third patient was a man, aged twenty-six years, of whom only X-ray films were shown. He had an atypical traumatic rupture of the left hemidiaphragm. He had been admitted to the hospital semiconscious and moderately shocked, with a fractured left clavicle and head injuries. X-ray examination of the left clavicle disclosed a crack fracture of the axillary portion of the second rib without displacement. The patient was more shocked than would be expected from the injury, and there was poor movement in the left lower part of the chest. X-ray examination of the diaphragm showed paresis of the left hemidiaphragm due to phrenic nerve palsy. Ruptured diaphragm was suspected, both clinically and on the X-ray appearances, but only after repeated films in the next two or three hours was it clearly demonstrable that there was an intragastric gas bubble appearing above the paralysed diaphragm. Recovery of consciousness furthermore permitted the eliciting of marked tenderness in the left upper part of the abdomen. After resuscitation an abdomino-thoracic incision was made, and a rent was seen extending forwards from the œsophageal opening close to the pericardium and involving the attachment at the diaphragm of the phrenic nerve. The nerve was severed at that point. The tear extended further forwards and to the left to within three inches of the diaphragmatic attachment to the eighth rib in the anterior axillary line. The abdomen contained a large amount of blood, whose origin was bleeding vessels in the diaphragm. The medial end of the tear was smooth, and there had obviously been a rupture from a preexistent hiatus hernia forwards over the dome of the diaphragm. Repair of the diaphragm was performed, the chest closed with drainage and the abdomen closed. Recovery was uneventful.

Dr. Reid said that traumatic rupture of the diaphragm was well known to resemble paresis of the diaphragm. It was usually not apparent until a variable interval after the injury. In the present case it was quite obvious within four to six hours of injury. Furthermore there was a tear of the phrenic nerve with paresis of the diaphragm as well as rupture of the diaphragm.

Carcinoma of the Common Bile Duct.

DR. ROWAN WEBB showed a married woman patient, aged fifty-six years, who had presented in March, 1955, with a three-months history of painless, progressive, obstructive jaundice, with a febrile onset, suggestive of infective hepatitis. On examination the patient was found to be deeply jaundiced, and both the liver and gall-bladder were palpably enlarged. The spleen was not palpable. Biochemical tests confirmed the clinical diagnosis of obstructive rather than infective jaundice. Laparotomy in April, 1955, revealed a localized tumour at the ampulla of Vater, completely obstructing the flow of bile into the duodenum. Resection of the head and neck of the pancreas and of the duodenum was performed, and end-to-end anastomosis of the stomach and jejunum restored intestinal continuity. The pancreatic duct, which was very dilated, was anastomosed to the side

of the jejunum, with embedding of the body of the pancreas in the jejunal wall, and an end-to-side anastomosis of the common duct to the jejunum was made. The operation site was drained, and a cholecystostomy was established. The patient made a good recovery, and since operation had remained well, and free from jaundice and from any symptoms of cholangitis. Microscopic examination of the specimen by Dr. J. Funder revealed a well-differentiated carcinoma of the common bile duct, with early local infiltration. No metastases were demonstrable.

Pancreatico-Duodenectomy for Chronic Penetrating Pyloro-Duodenal Ulcer.

DR. CONRAD LEY showed a married woman, who had been admitted to hospital on December 28, 1955, complaining of pain under the right costal margin associated with pain in the back and right scapular regions, not associated with nausea or vomiting, but associated with flatulent dyspepsia. Murphy's sign was present. Boas's sign was absent. No mass or viscus was felt in the abdomen. A plain X-ray film of the abdomen showed several stones in the vicinity of the gall-bladder. Normal findings were obtained from fractional test meal examination. The hæmoglobin value was 50%. The patient was prepared for laparotomy. At operation a large mass was found in the pyloric region, adherent to liver and pancreas, with enlarged lymph nodes. It was doubtful whether this was malignant or benign. Cholecystectomy and pancreatico-duodenectomy were carried out. The specimen removed consisted of stomach and seven centimetres of duodenum. On the lesser curvature and bordering the pylorus was a large round deep ulcer four centimetres in diameter. The edges were slightly undermined; the floor had a granular appearance and contained a small artery. Attached to the peritoneal surface was a large lymph node 2·5 centimetres in diameter. Microscopic examination showed the typical structure of a chronic peptic ulcer, but no changes suggestive of malignancy and no infiltration of stomach wall or ulcer floor by malignant cells. The diagnosis was chronic peptic ulcer.

Post-operatively the patient was surprisingly well, apart from some initial biliary drainage and severe pancreatic pain. Dr. Ley said that, despite the aids for investigation, the surgeon might be faced with the problem of a very large perigastric mass, either benign or malignant, and had to proceed with radical excision. Often the greatest help came from a carefully taken history. In the present case reward had come from the patient's being changed over from a life of chronic invalidism to a reasonably normal life.

Car Driver's Mangled Protruding Right Elbow.

As one of a series illustrating the principles of trauma in surgery, Dr. Ley showed a man, aged sixty years, who five months previously, when driving a truck with the usual protruding elbow, had been caught in a smash. The elbow was severely mangled, and the right clavicle was fractured. Examination showed a severely disorganized forearm, a badly fragmented proximal upper third of the ulna, a fractured middle third of the radius and fragmentation of the humeral condyles. Inspection showed that the arterial supply was intact. The state of the ulnar nerve was doubtful. Operative procedure consisted mainly of *débridement* (of soft and bony tissue) and immediate skin replacement in the degloved area. A Kirschner wire through the radius was brought out through the lateral condyle. Further grafting was carried out for coverage and removal of sequestra. Finally the nerves were shown to be intact.

In discussion, Dr. Ley referred to the importance of immediate handling and to the present functional state of the damaged limb. The patient had returned to work. He had fairly reasonable movement in his false joint, but no supination or pronation. He would be fitted with a small elbow splint to prevent lateral joint wobble.

Allergy Demonstration.

Members of the allergy clinic gave the following demonstrations.

DR. P. WARD FARMER showed a male patient, aged seventeen years, who had been stung three times by bees, each reaction being more severe than the one before. The last sting had produced an immediate reaction with extreme giant urticaria and bronchospasm. Desensitization was being carried out with a water-soluble extract of whole ground-up bee, and also with a bee venom extract. Dr. Farmer said that it was important to carry out desensitization over a long period, perhaps three years. Bee venom contained three main toxic factors, a hæmolytic factor, a neurotoxic factor and histamine, but the protein fraction must be minute in each sting. The sensitizing antigen seemed to be contained in the entire body of the bee and was independent of pollen carried by the bee, but the venom carried a minute amount of antigen peculiar to the bee's body. Hypersensitivity could occur without a history of previous stings, but in all fatal cases the subject seemed to have been stung previously. Probably the antigen of the bee, wasp and ant was the same or very similar, and the sensitizing sting might be that of a wasp or ant. When the sting was driven in, the barbs anchored it firmly; and as the bee escaped, the entire poison equipment, including the poison sac, might be torn free. Therefore the sting should be removed by careful "flecking" after separation of the poison sac.

The sensitive patient should always carry a strong antihistamine, preferably in liquid form, as that was absorbed more quickly than a tablet, and he should carry adrenaline and a syringe and be taught to give an injection to himself if stung. The use of oxygen and intravenously administered fluids was indicated as in the treatment of other anaphylactic reactions. In fatal cases death usually occurred within half an hour of the subject's being stung, and common symptoms were extreme giant urticaria, shock, cyanosis and respiratory difficulty. The commonest autopsy findings were pulmonary œdema and visceral congestion, but laryngeal œdema, pulmonary emphysema, cerebral œdema and cardiac dilatation might also be found.

DR. SAUL WIENER demonstrated some of the toxic and allergic actions of spider venom in laboratory animals. He showed live specimens of the red back spider (*Latrodectus hasseltii*), and photographs to illustrate the technique of dissecting venom glands of *L. hasseltii* and sections of the different types of venom glands found among spiders generally. He demonstrated that mice suffering from the toxic effects of spider venom displayed closure of the eyes, hypothermia and paralysis. A second group of mice which had received ACTH showed that that substance had no effect in reducing the toxicity of venom. Dr. Wiener said, however, that in view of the favourable reports concerning the use of cortisone in the case of a bite by the black widow spider (*Latrodectus mactans*), that substance was worth a trial in clinical treatment. Other measures for the treatment of arachnidism included intravenous administration of calcium gluconate and specific antivenene. Ampoules of red back spider antivenene were shown which Dr. Wiener had prepared at the Commonwealth Serum Laboratories. One millilitre of the serum neutralized the venom of about ten spiders.

Guinea-pigs were also shown which had become sensitized to spider venom and then died of anaphylaxis on further contact with spider venom. The post-mortem appearance showed gross emphysema of the lungs, which filled the thoracic cage. For comparison the thoracic contents of a normal guinea-pig were shown. It was pointed out that, apart from the toxic effects of the venom, allergic reactions might play a part in the symptomatology of arachnidism.

Asthma.

DR. S. BRAND showed eight patients with asthma who had been treated with cortisone and ACTH. He said that the indications followed were: (i) intractable *status asthmaticus*; (ii) intractable chronic asthma. (*a*) when no desensitization procedures were indicated, (*b*) when desensitization was proceeding, (*c*) when desensitization procedures had failed. In the first four cases, which were of indeterminate ætiology, the patients showed no skin sensitivities and all had nasal polypi. The condition had started in the third decade or later, and emphysema was a complicating factor in all. One patient was also aspirin sensitive. In the fifth and sixth cases the patient had failed to respond to desensitization, though skin tests showed well-marked reactions. The seventh case was complicated by repeated infections, though a definite allergic background was present. In the eighth case the patient had marked sensitivities, and desensitization therapy was in its early stages. Seven of the patients had had one or more admissions to hospital for *status asthmaticus*, and all had responded promptly to ACTH or cortisone after failure of adequate routine therapy. Relapse occurred in each case after cessation of hormone therapy. All were given maintenance therapy at one stage. The average dosage scheme was cortisone 25 to 100 milligrammes per day. Results were very difficult to assess. No patient remained free of asthma, and several needed admission to hospital for *status asthmaticus* whilst on maintenance doses. Generally, however, the frequency and severity of attacks and necessity for other drugs had diminished.

One patient, a Dutch girl, aged nineteen years, had had no idea that cortisone was a "wonder drug", and discontinued it at a level of 100 milligrammes per day on her own account whilst having severe asthma, saying that the tablets were doing her no good—she preferred "Ephedrobarbital". She was proceeding with desensitization, and was now much improved.

The patient in the seventh case, a girl, aged six years, was the only one who developed manifestations of overdosage (moon facies), and at that stage was admitted to hospital with *status asthmaticus*. She had been admitted on more than ten occasions. She now remained fairly well on a combination of maintenance cortisone and sulphonamide therapy.

Dr. Brand said that though there seemed to be no doubt of the usefulness of cortisone and ACTH in large doses in *status asthmaticus*, the results of maintenance therapy were far less certain at present-day dosages, but it did seem to have a definite place in treatment.

Psoriasis and Other Skin Diseases.

DR. IVAN WARTZKI showed patients and coloured transparencies illustrating psoriasis and its differential diagnosis. He pointed out that the sites of predilection of psoriasis were the extensor surfaces, especially the pressure points, such as elbows and knees, the scalp and the nails. He demonstrated that from the scalp the eruption sometimes extended onto the forehead to produce the *corona psoriatica*, the characteristic pitting of the nails resembling a ladies' thimble, the dystrophy of the nail plate and hyperkeratosis beneath the nail. Koebner's phenomenon (the presence of lesions along a scratch line) was also demonstrated, as was the absence of mucous membrane lesions.

Dr. Wartzki also demonstrated the flexural distribution of seborrhœic dermatitis and eczema, especially involving the natural folds (retroauricular, submammary, genito-crural and gluteal), in contradistinction to the distribution of psoriasis; the absence of Koebner's phenomenon and of mucous membrane lesions; and the association of seborrhœic dermatitis with *pityriasis capitis* (dandruff) and the presence of the *corona seborrhœica*. The character of the scales—dry, micaceous and silvery—in psoriasis compared with the greasy yellowish appearance in seborrhœic dermatitis was also demonstrated.

Lichen planus was shown to involve the flexor rather than the extensor surfaces, to consist of violaceous, polygonal, flat-topped papules with shining surfaces and milky white markings under the surface (Wickham's striæ), to exhibit Koebner's phenomenon, and to involve the buccal mucosa as milky white streaks. The absence of scales and the sparing of the scalp were also demonstrated.

Bowen's disease (multiple intraepidermal epitheliomata) affecting the covered parts was demonstrated; and the nodular edge to the lesions, differing from the previously discussed conditions, was pointed out. It was also mentioned that the condition sometimes coexisted with psoriasis, especially when the latter condition had been treated for some time with arsenic.

Other conditions of interest illustrated were multiple gummata, Boeck's sarcoid of the skin, *lupus vulgaris*, *pityriasis rosea*, *miliaria rubra*, *pityriasis versicolor*, parapsoriasis and the toxic erythematous macular eruption known as *erythema annulare centrifugum* (Darier).

Arthritis.

DR. GEORGE MCEWEN showed three patients suffering from arthritis.

The first, a woman, aged sixty-five years, had had rheumatoid arthritis for thirty-five years, presenting most of the characteristics of rheumatoid disease in its late stages and, after a progressively downhill course, emaciation, muscular atrophy, joint deformities, contracture and ankylosis. The patient was shown with the following thoughts in mind: (*a*) gross deformities in most arthritic patients could be prevented; (*b*) it was easier to prevent a deformity in arthritis than to correct one. The management of rheumatoid arthritis was discussed from the point of view of prevention of deformities.

The second patient, a man, aged fifty-three years, with rheumatoid arthritis, who had not responded satisfactorily to gold, "Butazolidin" or cortisone, had made a dramatic response to the new steroid prednisolone. Multiple rheumatic nodules, a feature of his case, were rapidly subsiding under prednisolone therapy. A section of a rheumatoid nodule from a biopsy of this patient was demonstrated.

The third patient was a man, aged thirty-three years, with polyarticular gout simulating rheumatoid arthritis and refractory to colchicine. Differential diagnosis and methods of treatment were discussed.

Neurological Conditions.

DR. A. C SCHWIEGER showed three patients with neurological conditions.

The first was a girl, aged seventeen years, whose vision had been found to be below standard at a routine examination. She herself had no symptoms, but her visual acuity was 6/18 right and left. Examination of the optic fundus showed bilateral healed macular chorio-retinitis, which was considered to be typical of the ophthalmological appearances in toxoplasmosis. X-ray examination of the skull revealed the presence of multiple linear calcifications, bilateral and scattered throughout the cortex and basal ganglia; some of them were of the "curvilinear" pattern stated to be typical of healed toxoplasmosis. Toxoplasma complement-fixation tests were carried out upon the patient and her mother. The patient's serum at a dilution of 1:20 fixed complement, and the mother's serum at a dilution of 1:10. The tests were carried out by Dr. E. French. It was assumed that the patient showed evidence of having acquired her infection in intrauterine life, although no relevant history could be obtained from the mother. It was noteworthy that the patient showed only some of the many manifestations seen in congenital toxoplasmosis, and those were discussed.

The second patient was a man, aged fifty-eight years. Two years previously he had complained of pain in the left side of his head; he did not describe this as headache, but said that rather it was a definite local pain, and accompanying it he noted that his left eye was turning outwards with consequent diplopia. Within a short time he noticed some interference in the function of his right-sided limbs, the foot dragged, the right hand seemed to lose power, and the right side of his face drooped. Speech, however, remained intact, and there was no disturbance of sensation. Examination revealed an incomplete left third nerve palsy and a mild right hemiparesis; sensation was intact, as were his visual fields. He had no cranial bruit. His cerebro-spinal fluid contained 80 milligrammes of protein per 100 millilitres, no cells were present, and the pressure was normal. The result of a Wassermann test was negative. X-ray examination of the skull yielded negative findings. It was suggested that this picture of a Weber syndrome might be explained on the basis of an aneurysm pressing on the left third nerve and the left cerebral peduncle. Left carotid arteriography showed no abnormality; so vertebral arteriography was undertaken and revealed a large aneurysm at the junction of the posterior cerebral and posterior communicating arteries on the left side. It was thought that this aneurysm had incompletely filled, and a small amount of air was introduced into the interpeduncular region. By means of tomography it was possible to demonstrate a considerably larger shadow illustrating the size of the aneurysmal swelling. It was pointed out that aneurysms in that situation were distinctly uncommon, and it was thought that the opportunity provided by the demonstration to show such a well-defined clinical picture would be of value.

The last patient was a man, aged sixty years, whose history was one of intermittent episodes of weakness affecting his left side with recovery over a period of twelve months. Within the last few months, however, he had experienced some difficulty with vision, and he felt that his "left eye" might be defective, for he not infrequently bumped into objects on that side. A further episode of weakness had taken place, but without recovery. He had no headache. Examination showed him to have mild left hemiparesis with a cortical sensory deficit, particularly in his fingers; there was complete left homonymous hemianopia. He was normotensive, and his peripheral vessels were moderate for his age. Auscultation over the right eye, however, disclosed a loud bruit, and it was thought that an angiomatous condition was likely to be responsible for his state. X-ray examination of the skull revealed faint calcification over a wide area in the right occipito-parietal region, with a large vascular channel running towards it. Right carotid arteriography disclosed an enormous arteriovenous malformation in the right parieto-occipital region fed by a large middle cerebral vessel. Left-sided arteriography also disclosed that this vascular mass was fed by an enlarged left anterior cerebral vessel crossing the midline. The condition of cerebral angiomata and vascular malformations in general were discussed.

Dr. Schwieger said that the first and second patients had been shown with a view to illustrating the relationship

of physical signs to proved anatomical abnormalities, although it was agreed that in neither instance could anything but symptomatic treatment be given.

Treatment of Scar Deformity.

DR. G. S. GUNTER presented details of a series of cases to illustrate various methods employed in the treatment of scar deformity. The methods illustrated by the cases included full-thickness pedicle flaps, full-thickness Wolfe grafts, dermatome grafts and "z" rearrangement.

Treatment of Cancer.

DR. B. A. STOLL demonstrated the cosmetic results of X-ray treatment of skin cancer affecting the face. Even with large ulcers over one inch in diameter the results of well-planned radiotherapy were shown to be cosmetically satisfactory, in addition to the high cure rate. It was only when underlying bone or cartilage was involved that surgery was preferred. Several examples of successful results in the nasolabial region and inner canthus area were demonstrated. Those areas were best treated by X rays in spite of the irregularity of the skin surface in the vicinity. In addition, good results were obtained in cases of epitheliomatous ulceration of the eyelids with careful shielding of the globe with leadfoil. The cosmetic result was very good, epilation of the eyelid being the only complication. By judicious variation of the size of the treated field during treatment, the edge of the treated area could be "faded off" into the surrounding tissue.

The results of radiotherapy for epithelioma of the lower lip were also demonstrated. From the point of view of the cure rate, the surgical figures and radiotherapeutic figures were very similar, varying with most authors from 60% to 80% of five-year cures, although early cases yielded a 95% cure rate. The cosmetic results demonstrated from radiotherapy in the advanced cases were good, the tissue defect being less than that which would follow surgery. In the early lesions treated by radiotherapy the cosmetic result was almost perfect when well-planned X-ray therapy was used, and it had the advantage of treating effectively adjacent areas of pre-cancerous keratosis. In the early lesions treated by surgery there was a danger that the less experienced surgeon might not remove the recommended two centimetres margin of healthy tissue (Cade), in an attempt to avoid extensive tissue repair. The radiotherapist had no such problem in irradiating an adequate margin.

Radiographs were demonstrated of the results of hormone treatment of patients suffering from breast carcinoma with established pulmonary or pleural metastases. Resolution of lung metastases or pleural effusions for periods up to three years was shown to follow administration of stilbœstrol. In a large series, over one-third of the patients treated with stilbœstrol survived a year or more, whereas less than 10% survived that time in the untreated group. The primary growth and gland metastasis were shown to respond to female hormone, particularly in the older age groups, whereas in bone metastasis recalcification and relief of pain occurred at all ages.

Morbid Anatomy Demonstration.

DR. J. F. FUNDER demonstrated morbid anatomical specimens of the following conditions: adamantinoma of the mandible, thymic tumour excised from a patient suffering from *myasthenia gravis*, lymphosarcoma and mucoid adenocarcinoma of the small intestine, Krukenberg tumours of the ovary secondary to a small primary gastric carcinoma, "fibroma" of the visceral pleura and leiomyoma of the stomach. The demonstration included presentation of summaries of the clinical features of the patients and of photomicrographs illustrating the histological features of the specimens.

Vascular Diseases of the Kidney.

DR. R. DRUMMOND presented a demonstration of certain pathological aspects of vascular diseases affecting the kidney. The diseases were subdivided into three groups: (*a*) those affecting mainly the glomeruli, (*b*) those affecting the glomeruli, arterioles and arteries, (*c*) those affecting mainly the arterioles and arteries. A description was given of the microscopic findings in each disease, and photomicrographs were shown pointing out the essential pathological features. A macroscopic specimen demonstrating each disease was also shown.

Eye Conditions.

DR. W. D. COUNSELL and DR. GEOFFREY SERPELL showed a number of patients suffering from ophthalmic disorders.

Lymphosarcoma of the Orbit.

The first patient was a male, aged thirty-eight years, who had been first examined in September, 1955. He gave the history that his left eye had been prominent for four years, and he then complained of a heavy feeling and pain in and about the orbit; before the vision failed in that eye, he had experienced diplopia. Examination of the patient revealed gross left proptosis with greatly impaired movements of the eye in all directions. The conjunctiva was œdematous with much dilation of vessels. There was pronounced œdema of the optic nerve head, visual acuity in the right eye being 6/6, and in the left eye 4/60. X-ray examination of the skull revealed rarefaction of the bone of the orbit on the left side, and there was no evidence of intracranial spread. The results of all other investigations were without significance. Immediately before operation the pain had greatly increased and the eye was blind. On November 18, 1955, left lateral orbitotomy was carried out, and it was found that almost all the orbital fat had been replaced by tumour; it was not possible to remove this and still leave the eyeball in place. In view of the operative findings, the pain, and the fact that the eye was blind, the orbit was exenterated and the cavity was grafted with split skin.

The comment was made that it was believed that the severe pain before operation was due to bone erosion by the tumour as shown radiographically and confirmed t operation, when the bone was seen to be very thin.

Dermatogenous Cataract.

The second patient was a male, who had had generalized eczema intermittently for twenty-two years; he had noticed a white spot in the right pupil one year previously. Examination of the patient revealed typical bilateral capsulo-cortical lens opacities which were demonstrated by slit-lamp microscopic examination.

Endocrine Cataract (Hypoparathyroidism).

The next patient was a female, who had hypoparathyroidism with tetany since a thyroidectomy eleven years before. Slit-lamp microscopic examination demonstrated typical bilateral cortical lens opacities of "endocrine type".

Paretic Strabismus.

A series of patients were demonstrated to illustrate the pre-operative management and post-operative results in seven cases of paretic squint of three ætiological types—traumatic squint, congenital squint, and squint following a cerebro-vascular episode. The importance of thorough orthoptic examination and repeated Hess-Lancaster screen studies at monthly intervals was demonstrated by charts for all patients. The comment was made that these patients demonstrated clearly the results which could be obtained from operation in cases of paretic squint. Three of the paretic squints followed head injuries.

Ophthalmic Sequelæ of Head Injuries.

A male patient, aged thirty years, who had sustained a severe head injury in April, 1955, was shown. His main complaint was of difficulty with reading, due mainly to right homonymous hemianopsia. In addition, signs of partial right-sided third nerve palsy could be demonstrated; they consisted in weakness of the right superior rectus and right inferior oblique muscles, weakness of accommodation in the right eye, and impairment of function of the efferent pupillary light reflex pathway on the right side.

SPECIAL GROUP ON AVIATION MEDICINE.

THE Special Group on Aviation Medicine (British Medical Association) will hold a scientific meeting at 49 Mathoura Road, Toorak, Victoria, on Wednesday, September 5, 1956, at 8 p.m. A talk on "Recent Trends in Pilot Selection, with Special Reference to North America" will be given by Mr. R. L. Want, Chief Psychologist, Royal Australian Air Force; this will be based on observations made during his recent visit to the United States of America and Canada. All members of the British Medical Association who are interested in aviation medicine are invited to attend this meeting.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

AN UNGRATEFUL CONVICT DOCTOR.[1]

[Governor Macquarie to Earl Bathurst, August 31, 1813.]

A PERSON named John Francis who was tried at the Middlesex Gaol Delivery on 10th July 1811, and Sentenced to Transportation for Life, and who arrived by the Ship Indefatigable at the Derwent in October last, being strongly recommended to me from Home, I was anxious to render his Situation as little irksome to him as the Nature of it would admit, and in Consequence I had him brought to Head Quarters from the Derwent, that I might have a personal Opportunity of Observing his Conduct, and extending him such Relief as he should appear to merit. Being bred a Surgeon, I had him employed in the General Hospital here as an Assistant, which is in itself an easy and relatively very comfortable Situation. Scarcely had this man been thus relieved from the Difficulties to which he was Subjected by his former Crime, than he effected his Escape from hence on board a ship called the Hope which sailed from hence on the 1st July last bound for the Cape of Good Hope. If this man should have the Temerity to proceed to England, I would hope your Lordship would cause him to be apprehended and returned hither, to undergo the full Measure of that Sentence, for the Relaxation of which he has already made so ill a Return by his Flight.

Correspondence.

THE TRIPLE SYNDROME OF BEHCET.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA of March 24, 1956, Dr. E. G. Saint and Dr. R. G. Linton report the rare case of Behcet's syndrome.

For the last six months this patient has been off all medical treatment. As she was still not feeling the best, she consulted a chiropractor who gave her massage, and a dietitian who put her on a special diet, which includes an excess of fruit and vegetable juices. On this she looks and feels better.

It would be appreciated if any of your learned readers can explain this phenomenon for me.

Yours, etc.,

A. BRECKLER, M.B., B.S. (Melbourne).

Mundaring,
Western Australia,
July 15, 1956.

THE PAIN OF PEPTIC ULCERATION.

SIR: In the issue dated July 14, 1956, of THE MEDICAL JOURNAL OF AUSTRALIA on page 71 appears your editorial "The Pain of Peptic Ulceration".

The pain of an internal ulcer may safely be presumed to be due essentially to the same thing as the pain of an external ulcer—the irritation of a nerve in some part of its path. Fortunately every doctor sees external ulcers of the lower legs. Every word and phrase you use in describing "the pain of peptic ulceration" can be applied to external ulcers of the lower legs. "A large ulcer may be painless and a small one may be excruciatingly painful. Pain disappears at the start of healing, though the ulcer is still present. . . . The pain in most cases was not related to the presence or absence of local free acid. . . . In only two patients did pain appear to be sometimes associated with local motor activity, and in the rest there was no correlation whatsoever. . . . It is clear that motility is not a cause of the pain. Free acid is associated with the pain, but only in some patients, and pain may occur in the absence of any free acid. . . . Every possible combination of local conditions was associated with pain or its absence."

Like the gastric juice, the sweat is acid. Like gastric ulcers, not all lower leg ulcers are excruciatingly painful. What causes the difference—which is very marked—in the severity of pain in lower leg ulcers? One thing, and only one thing, causes the severity of the pain—exposure of a small nerve trunk in the base of the ulcer. The most excruciatingly painful lower leg ulcer is a small ulcer, perhaps about the size of 3d., on the outer aspect of the fibular malleolus, with a small nerve trunk so bared and exposed that it can be seen. As of the peptic ulcer it is truthfully said "pain disappears at the start of healing" though the ulcer is still present. As soon as sufficient granulation healing tissue has formed to satisfactorily cover the small nerve trunk, the patient is relieved of the excruciating pain, although healing of the ulcer may take several days yet.

A woman, aged fifty-eight years, came to me on account of the extreme pain present in such a small ulcer over the malleolus of the fibula. The small nerve trunk could easily be seen running vertically in the base of the ulcer. She said: "The only relief I get is bathing it every two hours night and day." When advised to go to hospital and the dressing would be done by me very soon, and not for two days, she nearly fainted, repeating her statement it needed dressing every two hours. Before the two days were up, her excruciating pain had gone, and at the dressing the small nerve trunk was well covered with granulation tissue, and could not be seen.

Therefore "the pain of peptic ulceration" is explained by the simple fact that a small nerve trunk is bared at the base of the ulcer, and is irritated by the acid gastric juice and the inflammatory products present in the ulcer. Thus relief—partial and temporary—can be obtained by alkalis and food. Permanent complete relief comes with healing progressing to permanently cover the small nerve trunk, such healing going on to complete healing of the ulcer.

Yours, etc.,

LEONARD L. SNOW, M.B., Ch.M.

"Harley",
Inverell,
New South Wales.
July 20, 1956.

CAUDAL ANALGESIA IN LABOUR WARD PROCEDURES.

SIR: Dr. Maurice Barrett is to be congratulated on bringing to notice his experience with caudal analgesia in obstetrics (M. J. AUSTRALIA, July 7, 1956). My experience with this technique over the past four years has been confined to gynæcological and general surgical procedures involving the pelvis and perineum, when easy identification of the sacral hiatus is possible with the patient lying prone. I prefer to use a malleable caudal needle which virtually eliminates the catastrophe of breakage *in situ*, and favour a solution of 1·5% lignocaine with adrenaline 1:500,000, using 25 to 30 millilitres of solution. The height of analgesia obtained depends upon the balance between the volume of solution injected and the rate of escape into the paravertebral spaces, consequently it is unpredictable; I have never been convinced that gravity plays any role of importance.

Dr. Barrett has rightly been perturbed by a solution containing a concentration of 1:200,000 adrenaline, but I would suggest that the "muscular twitchings" he observed in six cases and the state of collapse in another were due solely to massive absorption of "Xylocaine" into the circulation from inadvertent intravenous injection, and not due to adrenaline toxicity as he suggests. I fail to understand the mechanism of his theory of "raised intracranial pressure by too rapid injection of solution" causing this phenomenon. To avoid this serious complication, frequent aspirations should be made during the course of injection of the anæsthetic solution, and this is especially important during pregnancy, when the sacral canal shares in the increased pelvic vascularity. Muscular twitchings can be followed by generalized convulsions, respiratory arrest, profound hypotension and death, so that immediate treatment must be given—that is, intermittent positive pressure respiration with oxygen, intravenous thiopentone, 50 to 150 milligrammes to control the convulsions (a larger dose only further depresses the respiratory and vasomotor centres), and a vasopressor drug such as "Methedrine" to combat the hypo-

[1] From the original in the Mitchell Library, Sydney.

tension. Such apparatus and drugs must always be ready when extensive local anæsthetic techniques are employed in the conscious patient, even though they may have had premedication with a barbiturate.

Yours, etc.,

OWEN MORGAN, F.F.A., R.C.S. (England).

7 Malcolm Street,
Perth,
July 18, 1956.

THE WONDER DRUGS OF PSYCHIATRY.

SIR: Dr. Godfrey Harris (M. J. AUSTRALIA, July 14, 1956) seems mainly to imply that modern drugs will cost a million pounds per four chronic patients. His reckoning is awry, at least as regards his postulates. He might as well calculate the cost of ACTH for an incurable arthritic on the basis of initial dosage. He fails to allow for the following:

1. The "usual" run of patients in mental hospitals (no longer "asylums") are not "lifers" now.

2. An object of the psychiatric drugs is to lessen the number of such life sentences. This they seem to achieve. If the average length of stay of patients so treated is 90% less, the stated cost would be justified, assuming that adjuvant costs average £3333⅓ per case treated, and taking Dr. Dax's figures, quoted by Dr. Harris, as correct. This is apart from incidental savings in breakages, destroyed clothes, other sedatives, shock treatment, and incalculable loss of efficiency in staff, patients and relatives, all of which have to be paid for by the State eventually.

3. The majority of patients will respond to the maximum dosage of chlorpromazine quoted by Dr. Morgan in three weeks, at an average cost (which a patient of mine pays, retail, for 25-milligramme tablets as 100 milligrammes are not available here) of 15s. per day, about one-twentieth of Dr. Harris's estimate. Perhaps the drug houses know where he gets his figures, but I, for one, do not intend to ask them.

Yours, etc.,

DOUGLAS EVERINGHAM.

21 East Street,
Rockhampton,
Queensland.
July 19, 1956.

SOME FACTS ABOUT MEDICAL REGISTRATION.

SIR: One point in your leader on medical registration (M. J. AUSTRALIA, July 21, 1956) needs correction. The imposition of an annual registration fee was indeed unpopular, but it is hard to believe that many members of an intellectual profession trained in precision of thought held that "as there had always been no annual registration fee, there should never be one". This verbatim quotation is an obvious *non sequitur*, clumsily expressed. Nor was it held that doctors on the register should not be required to pay for its proper upkeep.

The complaint, which will remain *sine die* until the cause is removed, is that the State, whatever its de-facto power, has no authority *de jure* to perpetrate injustice. A doctor already permanently registered must not be removed except for good cause. Good cause is not established by failure to pay a sum of money, payment of which was not part of the original contract between the parties.

The State has no more right to deal in dishonour than the persons who comprise it; they and the State are bound to keep good faith with those to whom they are obliged. The State could properly require an annual registration fee from persons not already on the register who subsequently seek registration. The State could justly require doctors already on the register to pay an annual sum towards its upkeep, proceeding in law to recover such a sum with damages and costs if it was unpaid; but it would have to show that the amount claimed was a just one, meeting without unreasonable surplus the purpose for which it was claimed.

Permanent registration may have been the deciding factor in settling some doctors in the State. It does our journal and Association little credit to say in effect, "serves them right; they should have found out about New South Wales State policies". The price of sound democracy is vigilance. The end of this particular wedge may be paper-thin, but at the other lie the evils which brought about the Nuremberg trials, and which are now part of the daily existence in those countries where atheism and contempt for individual human rights form the fabric of their rule.

Yours, etc.,

DOUGLAS VANN.

Mary Street,
Berridale,
New South Wales.
July 31, 1956.

THE MEDICAL BENEFITS FUND OF AUSTRALIA AND SERVICES RENDERED IN PUBLIC WARDS OF HOSPITALS.

SIR: I was interested in Dr. Grieve's remarks in THE MEDICAL JOURNAL OF AUSTRALIA, dated July 14, 1956, that the Commonwealth will not permit medical benefits to be paid for services rendered in public wards of hospitals.

The Commonwealth's instruction to organizations is that Commonwealth benefit must not be paid where a medical practitioner charges a fee in contravention of a State law. The Commonwealth cannot condone breaches of State law—hence, its refusal to pay Commonwealth benefit in such cases.

The organizations, however, are not prohibited from paying fund benefits. They are free to determine their own policy in such matters.

Yours, etc.,

A. J. METCALFE,
Director-General of Health.

Department of Health,
Canberra,
July 27, 1956.

SIR: Dr. Grieve's letter (M. J. AUSTRALIA, July 14, 1956) clarifies a matter which has long concerned general practitioners, especially in the country areas.

All gladly give their services to those in need, but it is unjust that patients who can and would repay are prevented from doing so whilst at the same time they are compelled to contribute so heavily towards their beds in a "public" ward. Injustice is the greater when a patient has specifically insured against certain contingencies and is prevented by apparent legalisms from securing equity.

This cannot be allowed to continue. Now that the approved societies have shown themselves powerless, unless our Association takes up the cause with resolution, the initiative will pass to the doctors most concerned. One scheme already under examination would give patients the benefits they usually require at a competitive figure, through groups of doctors acting as their own insurers. The premiums so deflected from the present approved societies, coming mainly from the country, should not materially affect their position. Inevitably, certain features of contract practice would have to be adopted. This could cause conflict and the evolution of a new organizing body more zealous for the country patients and the doctors who care for them.

With pronouncements of "official" medical policy so often opposed to the opinions of many members, it suggests that the Federal and State Governments are sometimes being misled over views held by a large proportion of the profession. It is important that these things should not be, and those in executive positions must more closely reflect the general view of members than support for the ministerial policy of the moment. This can be assured by more frequent reference of policy matters to local associations and closer attention to the resolutions of those bodies.

One such matter is the raising of British Medical Association subscriptions by 22%. This should not pass by a minority decision at the extraordinary general meeting on September 6, where only relatively few members can be physically present, unless the case is fully substantiated. So far, we have not been given a carefully considered statement by the Executive showing that all possible economies have been made and the reasons why such a rise is needed. For many doctors, whatever their gross takings may be, eleven guineas is a large fraction of the weekly net income.

The Association's professional and post-graduate work is good; it should not be offset by weakness in the medico-political and economic sphere. Too many of us can but rarely avail ourselves of the former and are compelled to endure the latter.

Unless the Executive recognizes these facts, it could come to face a declining membership. Even years of traditional loyalty to the Association will not withstand increasing economic pressure and disregard of many members' legitimate interests.

Yours, etc.,

Mary Street,
Berridale,
New South Wales.
August 4, 1956.

DOUGLAS VANN.

Post-Graduate Work.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR SEPTEMBER, 1956.

Refresher Courses for General Practitioners.

Gynæcology and Obstetrics.—A refresher course in gynæcology and obstetrics will be held at the Royal Women's Hospital, Carlton, from September 3 to 14, inclusive. Lectures and demonstrations will be conducted daily from 9.30 a.m. till 5 p.m. by the Clinical School of the hospital, with Professor Andrew Claye, of Leeds. A detailed timetable is available from the Melbourne Medical Post-Graduate Committee, with which enrolments should be made before August 20. The fee for the course is £14 14s.

General Medicine and Surgery.—A refresher course in general medicine and surgery will be held as follows:

Monday, September 17, at the Royal Melbourne Hospital, in the Lecture Theatre: 9.30 a.m., "Hypertension", Dr. J. L. Frew; 11 a.m., ward round, Professor Lovell; 2 p.m., "Surgical Conditions of the Large Bowel and Rectum", Dr. E. S. R. Hughes; 3.30 p.m., "Dermatological Problems in General Practice", Dr. I. O. Stahle.

Tuesday, September 18, at Saint Vincent's Hospital, in the Pathology Lecture Theatre: 9.30 a.m., "Diabetes in General Practice", Dr. W. Hamilton Smith; 11 a.m., "Cardiology", Dr. Maurice Clarke; 2 p.m., "Backache", Dr. T. King; 3.30 p.m., "Surgical Emergencies', Dr. J. L. Connell.

Wednesday, September 19: 9.30 a.m. to 12.30 p.m., at Fairfield Infectious Diseases Hospital, demonstration arranged by the staff of the hospital, followed by buffet lunch; 2 to 5 p.m., at the Royal Children's Hospital, in the Medical Lecture Room, demonstration arranged by the staff of the hospital; 7.30 to 9.30 p.m., at the Eye and Ear Hospital, demonstration arranged by the staff of the hospital.

Thursday, September 20, at Prince Henry's Hospital: 9.30 a.m., "Infection of the Hands and Feet", Dr. Rowan Webb; 11 a.m., "Treatment of Apoplexy", Dr. J. B. Curtis; 2 p.m., "Use and Abuse of Antibiotics", Dr. Ian Jones; 3.30 p.m., ward round or lecture demonstration, Dr. Theo Frank.

Friday, September 21, at the Alfred Hospital, commencing in the fifth floor Lecture Room: 9.30 a.m., "Diarrhœa", Dr. R. R. Andrew; 11 a.m., surgical ward round, Professor M. Ewing; 2 p.m., "Common Neurological Problems", Dr. J. Game; 3.30 p.m., "Surgical Conditions of the Stomach and Gall-Bladder", Dr. A. Rowlands.

Lunch each day will be provided by the hospital concerned or will be available in the hospital cafeteria. The fee for the course is £9 9s., and enrolments should be received by the Post-Graduate Committee by September 3.

Overseas Lecturers.

Dr. J. H. Sheldon's programme, commencing in August, will continue with lectures in the Medical Society Hall at 8.15 p.m. on Monday, September 3, and Thursday, September 6, on "Obesity, with Particular Reference to Maternal Obesity", and "Collagen Diseases" respectively. This will correct an error in our Diary Card Number 9 sent to annual subscribers, which stated that the lecture on collagen diseasec would be held on September 5. This lecture will be held on Thursday, September 6.

Professor Andrew Claye's programme as guest professor of the Royal Women's Hospital will continue on September 3, 4 and 6. In addition he will also take part in the gynæcology and obstetrics refresher course as mentioned above.

Country Courses.

Mildura.—At Mildura, on Saturday and Sunday, September 15 and 16, the following course will be given: Saturday, 2.30 p.m., "Head Injuries", Dr. J. B. Curtis; 4 p.m., "Diagnosis and Management of Abdominal Pain", Dr. Peter Parsons. Sunday, 9.30 a.m., "The Management of Diabetes", Dr. J. Stawell; 11 a.m., "Infertility", Dr. G. Ley. Dr. C. Bowman, 143 Deakin Avenue, Mildura, is the secretary of the Mildura Base Hospital Clinical Society.

Portland.—At Portland, on September 29, the following course will be conducted: "The Management of Arthritis", Dr. R. Strang; "Peripheral Vascular Disease", Dr. S. F. Reid. Dr. W. R. Angus, 214 Koroit Street, Warrnambool, is the secretary for this course.

Fees.—Fees for country courses are at the rate of 15s. per lecture, but those who have paid an annual subscription to the committee are invited to attend without further charge.

Flinders Naval Depot.—At Flinders Naval Depot, on Wednesday, September 12, at 2.30 p.m., Dr. B. Keon-Cohen will conduct a demonstration on an orthopædic subject, by arrangement with the Royal Australian Navy.

Course in Neuropathology.

At 7.30 p.m., on September 4, 6, 11 and 13, Dr. Ross Anderson will conduct a course in neuropathology suitable for candidates for the D.P.M., Part II. The fee for this course will be £3 3s., and enrolments should be made with the committee by August 21.

Postponement.

The course arranged for August 11 at Swan Hill has been postponed till October 27, 1956.

Inquiries.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

Obituary.

HENRY WILFRED PALMER.

DR. HENRY WILFRED PALMER, whose death was announced recently in these pages, was born at Burwood, New South Wales, on May 26, 1877. He was the second son of Edward Gillett Worcester Palmer and began his education at Burwood Public School and the Sydney Boys' High School. He left school at fifteen years of age and went to Hazelbrook on the Blue Mountains and worked on the family orchard at the place where the R. T. Hall Sanatorium now stands. He lived at Hazelbrook for seven years, fruit growing and road contracting. When his brother Charles Reginald decided to study medicine, Henry made up his mind to do the same. They became medical students at the University of Sydney and completed the course together, graduating in 1906. After a short period spent at the Queen Victoria Sanatoria at Wentworth Falls and Thirlmere, Palmer joined the New South Wales Public Service in June, 1907, and served as resident medical officer at the Coast Hospital. He resigned in August, 1908. In April, 1909, he was appointed first medical superintendent of the Waterfall Sanatorium under the Metropolitan Hospital and Charities Department. He again became an officer of the Department of Public Health when the Waterfall Sanatorium was transferred to it on March 1, 1913. Palmer guided the destinies of the Waterfall Sanatorium as medical superintendent for thirty years, retiring in 1939. During this time he was recognized as an authority on pulmonary tuberculosis; his opinion was always available to, and valued by, the Editor of this journal. In 1924 he visited and reported upon the sanatoria of New Zealand, Victoria and South Australia, the report being published with the papers of the Legislative Assembly for that year. From 1939 to 1955 Palmer was the Secretary and Chief Executive Officer of the Carrington Convalescent Hospital at Camden. During his 46 years of hospital administration he served on many committees, boards and advisory panels for the Government.

Palmer's main hobby was gardening, and he was an active member of the Western Suburbs Flower Club. He was interested all his life in farming, and especially in pig-

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 28, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1(1)	5(5)	6(5)	..	1(1)	..	..	..	13
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	3	..	1	..	..	..	..	..	4
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(1)	14(12)	..	..	1	..	..	..	19
Diphtheria	4(2)	1(1)	..	..	1	1	..	..	7
Dysentery (Bacillary)	..	1(1)	..	3(3)	..	..	..	..	4
Encephalitis	2	..	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	49( )	48(21)	..	10(7)	4(1)	7(3)	3	1	122
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	4	..	..	..	..	..	4
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(1)	4(2)	2	1(1)	1(1)	5	..	..	16
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2	1(1)	1	..	..	6(3)	..	..	10
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	17(16)	..	..	2(2)	..	..	..	19
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	8(4)	16(10)	5(2)	6(3)	2(2)	4	..	..	41
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	4(1)	..	..	..	..	..	4
Trachoma	..	..	..	..	11(6)	..	..	..	11
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	24(20)	32(22)	16(11)	9(6)	6(5)	5(3)	1	..	93
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	2(1)	..	..	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 4, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	2(1)	1(1)	..	1	..	..	..	5
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	2	..	13	..	..	..	..	..	15
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1(1)	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(1)	9(8)	1(1)	..	1(1)	..	..	..	13
Diphtheria	2(1)	1(1)	..	..	2(2)	..	..	..	5
Dysentery (Bacillary)	..	..	2	1(1)	..	..	..	..	3
Encephalitis	..	1(1)	..	2(1)	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	69(27)	53(24)	..	10(6)	4(1)	7(1)	2	2	147
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	15	..	..	..	..	..	15
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(1)	4(1)	..	..	..	1	..	..	8
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	1(1)	..	..	..	..	..	..	1
Paratyphoid	1	..	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1(1)	5(3)	1(1)	1(1)	..	2(1)	..	..	10
Puerperal Fever	1	..	..	..	..	..	1	..	2
Rubella	..	16(11)	..	..	1(1)	..	..	..	17
Salmonella Infection	..	..	..	1	..	..	..	..	1
Scarlet Fever	14(9)	..	5(2)	3(3)	1	..	..	..	23
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	28(15)	19(14)	11(7)	9(5)	13(9)	5(2)	..	1	86
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	2(2)	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

raising. He pursued the latter at both the Waterfall and Carrington Hospitals where it developed into a successful side-line. Palmer is survived by his widow, one son and two daughters. He was one of those whose names do not appear in newspaper headlines, but who constitute the backbone of the profession.

Royal Australasian College of Surgeons.

NEW SOUTH WALES MEETING.

A MEETING of the Royal Australasian College of Surgeons will be held at the Stawell Hall, 145 Macquarie Street, Sydney, on August 22, 1956, at 8.15 p.m. The subject will be "Multiple Renal Calculi", and the speakers will be Dr. H. Pearson and Dr. J. McNamara. All medical practitioners are invited to be present.

The Royal Australasian College of Physicians.

APPLICATIONS FOR RESEARCH GRANTS.

APPLICATIONS for grants for medical research for the year commencing January 1, 1957, will be considered by the Research Advisory Committee of The Royal Australasian College of Physicians in September next, and grants will be made by the Council at its meeting in October. Information regarding the conditions of award of research grants or grants-in-aid is available on application to the Honorary Secretary of the College, 145 Macquarie Street, Sydney. The closing date for the receipt of applications will be Friday, September 7, 1956.

Notice.

THE CIBA FOUNDATION AWARDS.

A PANEL consisting of Professor C. H. Best (Toronto), Professor E. Braun-Menendez (Buenos Aires), Dr. G. W. Corner (New York), Professor A. Haddow (London), Professor R. Nicolaysen (Oslo), Dr. A. S. Parkes (London), Professor F. Verzar (Basle) and Professor F. G. Young (Cambridge), with Professor E. J. Conway (Dublin) and Professor V. R. Khanolkar (Bombay) as corresponding members, has considered 75 papers from 17 countries for the Ciba Foundation's awards for 1956 for research relevant to the problems of aging, and the following awards have been made (the names of leading authors only are given):

Madeline Keech and R. Reed (jointly) (University of Leeds), £400; B. Bronte-Stewart (University of Cape Town) and R. L. Gregory (University of Cambridge), each £300; J. Conway (Charing Cross Hospital, London), £200; J. E. Birren (National Institute of Mental Health, Bethesda), L. A. F. Carlson (King Gustav V's Research Institute, Stockholm), G. H. A. Hevelke (Medical University Clinic, Leipzig), K. Schreier (University Clinic, Heidelberg) and C. A. Villee (Harvard Medical School, Boston), each £100.

The awards are being offered again in 1957, the closing date for entries being January 31, 1957, and further information may be obtained from the Ciba Foundation, London.

Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Domany, Susan, M.B., B.S., 1956 (Univ. Sydney); Hepburn, Susan Frances, M.B., B.S., 1956 (Univ. Sydney); Prosser, David John, M.B., B.S., 1956 (Univ. Sydney); Protopopoff, Serge Nicholas, M.B., B.S., 1956 (Univ. Sydney); Lang, Francis Houston, M.B., B.S., 1955 (Univ. Sydney); Rose, Michael John Asher, M.B., B.S., 1955 (Univ. Sydney); Arnott, Noel George, M.B., B.S., 1953 (Univ. Sydney); Feneley, William Henry, M.B., B.S., 1953 (Univ. Sydney); Finlayson, Jean Stanley, M.B., B.S., 1939 (Univ. Melbourne); King, Errol Edward Trevor, M.B., B.S., 1945 (Univ. Sydney); Rankin, James Gerald D'Arcy, M.B., B.S., 1954 (Univ. Sydney); Wansey, Richard Barry, M.B., B.S., 1954 (Univ. Sydney); Klavans, Arvis, licenced under Section 21 (b) of the *Medical Practitioners Act*, 1938-1955.

Corrigendum.

WE are informed that an error has occurred in the issue of August 4, 1956, under the caption "Australian Medical Board Proceedings" (New South Wales). The name of Dr. Allen Cutler Green was given as Allen Butler Green. The mistake was made in the *New South Wales Government Gazette*, Number 77, of July 13, 1956, from which the information was taken.

Deaths.

THE following deaths have been announced:

BALLANTINE.—Jane Margaret Ballantine, on August 8, at Sydney.

O'NEILL.—Vernard Francis Aloysius O'Neill, on August 3, 1956, at Townsville, Queensland.

BROTCHIE.—Edward Bonaventure Heffernan Brotchie, on August 13, at Kingsgrove, New South Wales.

HUDSON.—Charles Burgoyne Hudson, on August 15, 1956, at Sydney.

Diary for the Month.

AUG. 25.—Queensland Branch, B.M.A.: Annual General Meeting.
AUG. 27.—Victorian Branch, B.M.A.: Branch Meeting.
AUG. 28.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 30.—New South Wales Branch, B.M.A.: Branch Meeting.
SEPT. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.